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Symposium on Lung Tumors

Bronchogenic Carcinoma

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Lung cancer has become pandemic in North America and the industrial countries of northwestern Europe. It is increasing more rapidly than any other form of cancer. According to Ochsner,[1] if the present rate of increase continues, bronchogenic carcinoma will involve 47,000 persons in the United States by 1970. It has been estimated that approximately 21,000 died of lung cancer in 1952.[2] It is agreed that the increase is real and not the result of better reporting or examining or of an aging population.

INCIDENCE

According to the International Conference on Lung Cancer report in 1952,[3] mortality increased twenty-five times between 1914 and 1950. The relative increase has been five times greater for males than for females. Among men the increase in mortality has been 500 per cent in 20 years. Male deaths from lung cancer alone in 1952 exceeded deaths from tuberculosis.

According to Churchill,[4] the greatest incidence is in men between 40 and 70 years of age, the average being 55. Bishop[5] also found this to be true in 100 cases in New Haven Hospital. Youth, however, offers no immunity and definitely increases the hazards if the disease occurs.

The incidence is about the same in the white race and in the Negro race.[6] In the oriental races cancer of the lung is relatively rare, being eighteenth in order of frequency in a study of the Chinese.[7] In a discussion of their study, Yeh and Cowdry point out that the underdevelopment of thoracic surgery in China may be an explanation for this.

The right lung is more often involved than the left. In an unreported group of 100 cases at the New Haven Hospital the ratio was 53 to 45.[5] Kikuth's[8] studies revealed a ratio of 73 to 62.

Unlike cancer of the stomach and colon, which varies inversely with the economic status of the population, cancer of the lung knows no social or economic barriers.

ETIOLOGY

The causative factors of bronchogenic carcinoma are unknown. Because of increased and insistent interest in the subject, probable carcinogenic agents are being aggressively explored in this and many other countries. Owing to the acuteness of the problem, unproved statements are rampant. Leddy and Moersch,[9] in a study of 250 cases of lung cancer at the Mayo Clinic, concluded there are no tangible etiologic factors.

Workers in asbestos plants, in chemical plants where radioactive substances and gases are studied, handlers of lubricating oils and gasoline products, and workers dealing with arsenic, cobalt, or chromate products are considered to be in hazardous occupations. Industrial tars are thought to be responsible, but this has not been proved.

Inhalation smoking is at the moment under severe indictment as a cause of bronchogenic carcinoma. Many studies are being currently made in this area. A recent one by Watson and Conte[10] perhaps reflects opinions shared by many scientists. In a study of 769 consecutive patients at the Thoracic Clinic at Memorial Hospital, New York, 301 patients had histologically proved cancer of the lung. Of these 265 were males and 36 were females. Of the males 260, or approximately 98 per cent, were smokers, and 5, or approximately 2 per cent, were nonsmokers. Of the 36 female patients with cancer of the lung 21, or 58 per cent, were nonsmokers while 15, or 42 per cent, were smokers. The report published in England by Doll and Hill[11] parallels these figures rather closely.

Explanation for the disparity in the sexes has not been satisfactorily made. One possible explanation is that the bronchial mucosa of women is less susceptible to malignant changes than is that of men. Another is that women as a group have not smoked for as long as men have; that is, smoking among women is a relatively new development.

It is generally agreed that it is not definitely known if a carcinogen is present in tobacco. The arsenic content has aroused speculation, and Alvarez[12] and Regato[13] suspect the tar content, but neither the arsenic nor tar content has been proved to be carcinogenic. However, these and other possible etiologic factors show the necessity for competent and unbiased research into this critical problem. Various interested groups, such as the American Cancer Society[14] which in 1953 spent 5 millions of its approximately 20 millions in research on this problem, are already doing this.

ORIGIN AND SPREAD

According to Karsner and Saphir[15] there is no evidence that carcinoma of the lungs originates elsewhere than in the bronchi. Occurring near a bifurcation of a large bronchus, it spreads to the mediastinal and hilar lymph glands. The nature of the growth determines the course. Flat growths are symptomless early, while nodular formations may promptly fill the lumen and result in obstruction and consequent atelectasis. Upon perforation of the bronchial wall the tumor traverses the peribronchial lymphatics which accounts for the cuff and frazzled margin often observed on x-ray. On the radiating lymphatic strands nodules are often seen, even on the pleura. Less frequently the tumor mass forms in a distant bronchus of small dimensions. From this a radiating band usually connects with the hilum and may be radiographically revealed.

HISTOLOGY AND MORBID ANATOMY

Definite classification of various primary lung carcinomas is impractical if not impossible. Predominance of a specific type of cell, however, identifies the neoplasm. Three groups are usually recognized both in the United States and in Europe:[16] (1) squamous cell carcinoma, (2) columnar cell and spheroidal cell cancer (adenocarcinoma), and (3) anaplastic, oat cell, or round cell tumors.

Squamous cell cancer arises from the metaplastic bronchial epithelium and is composed of large cells which form a dense white mass in the lung root. Metastatic spread, small and discrete, is usually in the neighboring lymph glands. The average age of incidence is 46 years. This type accounts for 30 per cent of pulmonary cancer.

The columnar and spheroidal type (adenocarcinoma) arises in the mucous glands or bronchogenic epithelium. The large cells may be columnar or cuboidal, often with an alveolar arrangement. The less

the cells are differentiated the more they are disseminated. The average age of incidence is 51 years. Metastasis is extensive, spreading to practically all tissues with varied frequency. Twenty-five per cent of lung cancers are of this type.

In anaplastic or round-celled tumors the cells are smaller than in the preceding types, but the nuclei are large. Macroscopically these tumors resemble the columnar type from which distinction is difficult. In these neoplasms a well-developed vascular supply is found with predilection to degeneration. They are highly malignant and metastasize freely, simulating in this respect the columnar type. The average age of incidence is 43. Of all lung cancers, 40 per cent are of this type.

Knights[17] states that, in a review of more than 6,500 autopsies and a study of surgical specimens at the Henry Ford Hospital, the brain was found to be involved by various types of malignant tumors either by metastasis or extension in 94, 58 males and 36 females. In 109 cases of pulmonary carcinoma the brain was examined in 32; 17, 53 per cent, were found to have cerebral metastases. All types of pulmonary cancer were found.

SYMPTOMATOLOGY

The onset of bronchogenic carcinoma is usually insidious; discovery is often accidental in gastrointestinal studies or in mass chest surveys. The extent of the preclinical period is indefinite.

Frequently loss of weight and vitality with dyspepsia are initial signs. Symptoms referred to the chest, such as cough, pain, or blood-streaked sputum, may motivate the patient's first consultation with a physician. The symptom of a protracted recurring cold with variable fever may be misdiagnosed as chronic bronchitis. Symptoms of unresolved or virus pneumonia may prevail. In the aged, confusion with tuberculosis is not unknown.

Usually there is a cough, fairly dry at first. If brassy, the recurrent laryngeal nerve is involved. In the scant sputum, cancer cells may be present. If hemoptysis occurs it is usually slight and unpredictable. Pleurisy, dry or with blood-stained exudate, may occur.

Pain is variable, but is severe in 50 to 60 per cent of the cases.[3] It may be due to bone or pleural involvement, or, if in the arm, to an invasion of the bronchial plexus (Pancoast's syndrome).

Loss of weight and weakness signify a well-developed case, while a wheeze and dyspnea suggest bronchial obstruction, often not present in the parenchymatous type. Cyanosis suggests an advanced state. It may occur suddenly or gradually. Paroxysmal tachycardia is not unusual and is the result of enlarged mediastinal glands involving the sympathetic and vagus nerves. Pulmonary osteoarthropathy rarely occurs, owing to the brevity of the course.

The average duration of a case of cancer of the lung is less than 1 year. Malignancy varies inversely with the patient's age.[16]

DIAGNOSIS

There are no infallible roentgenologic signs of bronchogenic carcinoma. However, roentgenography is the best single means for its discovery. For example, 83 per cent or 310 were positively diagnosed by this means in a study by Ochsner[1] of 371 patients with bronchogenic carcinoma. Ochsner stated that atelectasis as the result of bronchial obstruction with obstruction distally in a man past 40 is indicative of bronchogenic carcinoma.

Early x-ray evidence includes unilateral hilar infiltration, a fan-shaped atelectatic area, a shift of the mediastinal structures to the affected side, and fixation of the hemidiaphragm.

In distal areas, bronchography is of value. Bronchoscopy, although of value, reveals only about one third of the cases of cancer.[1] Ochsner, DeBakey, and Dixon[18] performed primary pulmonary resection on 147 patients of 246 on whom exploratory operations had been performed. Bronchoscopy with biopsy was performed on all but 22 in whom the peripheral location of the tumor made the procedure impracticable. Seventy per cent of cases can be identified by cytologic studies.[1]

Malignant cells in the sputum, pleural effusions, or bronchial secretions are always confirmatory. Surgical removal of tissue for biopsy study is proper. Opposition to securing a specimen with the aspiration needle because of the fear of infecting traversed tissue seems to have sharply diminished.

Planigraphy, angiocardiography, and pulmonary angiography have found favor as diagnostic procedures. These methods enable the surgeon to evaluate the relationship of the neoplasm to the vascular structures in the operative procedure. Although experimental at present, supervoltage diagnostic roentgenography may become of value since the mediastinal structures are simultaneously included.

RADIOLOGIC CONSIDERATIONS

Radiologically speaking, bronchogenic cancer presents an ever-changing panorama, hence a serial roentgenographic study is indispensable. Specific classifications are elusive; even basic grouping presents practically insurmountable problems. However, many writers[5,16] recognize for convenience of discussion two types: (1) hilar or central, and (2) parenchymatous, lobar, or nodular.

As previously indicated, the site may be in a large central or a peripheral bronchus. If central, usually in the first section of a main bronchus, the hilar glands are rapidly infiltrated and major atelectasis of an entire lobe may be ushered in. In the radiograph the diffuse type is fan-shaped, spreading into lung tissue, has blurred hairy margins, and fades off at the periphery. In about 75 per cent of cases, bronchogenic carcinoma arises in a major bronchus.[19] Should a medium-sized bronchus be invaded, hilar involvement comes much later via lymph channels. In this instance, a fan-shaped area is presented with the apex toward the hilum, resembling a pneumonic consolidation. Pneumonitis often results.

ATELECTASIS

Atelectasis and bronchial emphysema result from bronchogenic carcinoma by one of two methods: either by obstruction or by compression of a bronchus. The one is from within, the other from without. Development may be gradual or precipitous. The atelectatic shadow may vary greatly in opacity and in size, for it may comprise a segment, a lobe, or an entire lung. The affected portion is concave toward the involved area; this is revealed by the interlobar fissure or a collapsing of the ribs, which narrows the intercostal spaces, and by elevation of the diaphragm.

BRONCHIECTATIC CAVITATIONS

Bronchiectatic cavitations follow bronchostenosis. They are found only in cancer of the larger bronchi. Fishberg and Rubin[20] found abscess formation in approximately one third of their cases. Cavitations usual-

ly occur in a sequential manner: bronchostenosis, atelectasis, infection, perforation of the bronchial wall with consequent abscess and cavity formation; or, as a reasonable probability, the tumor outgrows its own blood supply, and self-disintegration and destruction result. Cavitations appearing as translucent areas in a collapsed lobe may sometimes be observed.

METASTASIS

Metastasis occurs early with wide distribution by direct invasion of the vascular system or by lymphatic extension to other organs. According to Bishop,[5] if the regional nodes of the pulmonary hilus are included, true metastasis or lymphatic extension occurs in 80 to 95 per cent, which is greater than from any other viscus. Distribution is rapid and practically unlimited. It probably is aided by the mechanical factors of breathing, coughing, and sneezing which disseminate the cells by way of the numerous bronchial and vascular channels.

NODULAR TYPE

The nodular form arises from a small bronchus. It early penetrates the wall and grows as a rounded nodule maintaining its outline indefinitely. Approximately 25 per cent of bronchogenic cancer originates in a small bronchus and develops into the peripheral or nodular tumor.[19] Growth is rather rapid, and there is a definite increase in size within 1 to 2 months. The tumor is very malignant, terminating the life of its host within approximately 18 months; with the hilar type expectancy is greater than 2 years.[16]

The nodular type rarely crosses an interlobar fissure. Invasion is by means of the hilum as noted by a small infiltrating band uniting them. It usually remains single, while the sharp, well-defined margins are usually maintained. Atelectasis is not the rule but may result from compression of a bronchus.

Clinical signs are uncertain. Pain is constant but variable in degree. Dyspnea is not an early symptom and may be absent. Cough is variable. Evidence of the seriousness of the condition develops rapidly and unrelentingly.

With the above classification it must be recognized that every conceivable variation may occur. Bronchogenic carcinoma is always an outlaw in rich, unchartered, and largely unguarded territory, such as lung tissue.

SUPERIOR PULMONARY SULCUS TUMOR (PANCOAST'S TUMOR)

Pancoast's tumor is rather rare but is a well-known entity. Occurring high in the apex, usually the right, it involves mostly males. Hall[21] made a survey of the considerable number of cases reported and found that in 50 per cent of them the lesion was primary carcinoma, and the remainder were the result of metastases. The tumor most often affects middle-aged persons.

Symptomatology is definitely out of proportion to the radiologic findings. The tumor always occurs at the thoracic inlet and is revealed in the x-ray as a small homogeneous shadow. It early involves the brachial plexus, cervical sympathetics, and blood vessels, producing severe pain in the shoulder and arm and often atrophy of the painful areas. It frequently produces Horner's syndrome on the involved side. There is rib destruction at the site of involvement, and often there are spinal erosions.

The period of involvement is brief, a year or less. It is a strange paradox that death results from such a small tumor in which x-ray, instead of revealing metastases, shows only invasion.

Treatment usually, but not always, fails. Surgery and radiotherapy are seldom more than palliative. The tumor is considered radioresistant.

TREATMENT

X-ray therapeutic tests deserve consideration. By reducing the size of an endobronchial tumor an atelectatic condition may be temporarily relieved. Roentgenotherapy for this condition, if employed, must be adequately and decisively given.

Surgery is the treatment of choice of primary bronchogenic carcinoma, and pneumonectomy is the only worth-while surgical procedure. Cases are either operable or inoperable.

Some of the indications of inoperability are the presence of malignant cells in the pleural fluid, involvement of the phrenic nerve or brachial plexus, metastatic spread in distant areas, extension to lymph nodes, lymphatic dissemination, infiltration into adjacent organs and tissues,[4] bilateral involvement, and far advancement in age. Of 1,122 patients studied by Ochsner,[1] 43 per cent had inoperable lesions when first seen. Resection was performed on 33 per cent of the entire group. The hospital mortality rate for 372 cases in which resection was done was 20 per cent. Of those resected, 18 per cent were alive after 5 years. Of the 317 patients who were not resected, none lived longer than 4 years, only 1 that long. Studies by others essentially substantiate these figures. Delay is invariably fatal.

As a mode of treatment roentgenotherapy has a definite and important place. All cases of inoperable bronchogenic carcinoma should have routine irradiation. Certain types, such as the oat-celled tumor, are highly sensitive to irradiation; hence, good although temporary results are obtained.

Moderation of pain, drying up of excessive secretions, and easing of the many discomforts in the malignant chest render x-ray therapy an advantage not to be denied. It obviates the too frequent inhuman statement to the terrified terminal patient, "There is nothing more to be done." Being definitely palliative alone justifies its use.

SUMMARY

Of the various methods for recognizing lung cancer the roentgenogram is one of the most essential for screening purposes, being accurate in 83 per cent in one large series.

Confirmation is by identification of the carcinomatous cells in involved fluids or tissues.

Surgery is the first choice of treatment in operable cases.

Roentgenotherapy for palliation is indicated in all inoperable cases and for certain testing in the absence of surgery.

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The Present Status of and Recent Advances in the Roentgenology of Benign Tumors of the Lung

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The definitive diagnosis of benign pulmonary tumors, solely on the basis of their roentgenologic characteristics, currently rests on very insecure ground. The complacent attitude in the classification of these lesions in years past has been shaken into a startled awakening by the advances in the last decade in the allied specialty fields—bronchoscopy, anesthesiology, thoracic surgery, and histocytology. Nowadays it cannot confidently be asserted that any circumscribed pulmonary neoplasm is not potentially malignant, even though it may have been observed over a period of years and adjudged roentgenologically stable. One by one, the former criteria for benignancy of lung tumors have been questioned, re-evaluated in the light of new knowledge, and discarded or relegated to the limbo of "merely suggestive findings." It is, therefore, with a keen sense of personal inadequacy that I discuss the present status of and recent advances in the roentgenology of benign tumors of the lung.

The so-called true benign tumors of the lung are rather rare; they include the fibromas, myomas, osteomas, angiomas, lipomas, xanthomas, and adenomas, plus the mixed tumors of those origins. Additionally, in differential diagnosis a multitude of other neoplasms and pseudoneoplasms must be considered which include hamartomas, closed fluid-filled pulmonary cysts, the great variety of granulomas, encapsulated foreign bodies and encased lipids, residual closed pulmonary abscesses, unresolved consolidative bronchopneumonias, intrapulmonary thymomas, invaginated intrathoracic thyroid adenomas, pleural mesotheliomas that dip into the alveolar lung structure from the visceral layer, and even unusually configured pulmonary infarctions. Carcinomatous metastases can, all too frequently, be readily confused with benign tumor.

I have been impressed and alarmed by the changing clinical status of the coin lesions. The term "coin lesion" refers to those solitary, sharply defined, rounded zones of increased density that measure from 1 to 6 cm. in diameter, which are surrounded by normal lung tissue. Although a rather high percentage of coin lesions consists of tuberculomas, any of the above mentioned tumors may fall in this classification and present identical radiographic shadows. Those of tuberculous etiology, being relatively solid granulomas, tend to be firm, encapsulated, laminated, free of pericapsular inflammatory zones, and sometimes flecked with calcification, and often exhibit central caseation. They are commonly located in the upper segments of a lobe near the periphery. It is generally agreed that histopathologic examination should be made of most coin lesions obtained at exploratory thoracotomy. The incidence of malignancy in these tumors, which were considered benign in prior years, has averaged 15 to 30 per cent in recent reports of several series of cases appearing in the literature.[1]

Pulmonary hamartomas are overgrowths of bronchial tree elements; essentially they are cartilaginous, but exhibit admixtures with other differentiated cell growths. They are closely related to the pure chondromas. Though these lesions may become so large as to fill a hemithorax, they are usually less than 6 cm. in diameter and commonly are flecked with calcium throughout, which aids in distinguishing them from echinococcic cysts. The calcification pattern in the older echinococcic cysts is peripherally distributed in the capsule. These tumors are not prone to undergo malignant degeneration.

The lipomas, fibromas, and myomas occurring in the pulmonary structure present no distinguishing characteristics and may be accurately identified only after surgical exploration and pathologic study. Hemangiomas of the lung are quite rare, but may be diagnosed with some certainty by variations of the Valsalva maneuver, the tumor becoming smaller when the patient holds his breath.

I approach the controversial and complicated subject of bronchial adenomas with caution. It is common practice today to classify and discuss them with malignant lesions. Most chest specialists have had occasion to observe cases pronounced benign following bronchoscopy and biopsy which later demonstrated metastatic behavior.

Recent roentgenologic advances of value in a consideration of benign lung tumors include angiopneumography, the mapping of some intrathoracic tumors by scintillation counters following low millicurie isotope dosage, visualization of questionable lesions and the surrounding lung structures by use of the new fluoroscopic image amplifier, and further refinements and wider use of body section roentgenography.

Angiopneumography, utilizing one of the high iodine content dyes available for intravenous injection, usually by catheter technics and multiple rapid film exposures, is relatively new. It apparently is of most value in tumors of moderate or large dimensions.

Briefly, interpretation is based upon the impression that inflammatory lesions fail to create any arterial amputation or produce appreciable vascular malformation; malignant tumors demonstrate stenosis or arrest of arterial rami; benign neoplasms distort the arterial branches without occluding them and the neighboring arterial supply is unaltered. I have had no experience with such examinations. Vascular catheterization procedures with rapid injection of relatively large amounts of high iodine content dyes produce a very definite percentage of morbidity and mortality, possibly higher than that of exploratory thoracotomy per se to which most cases will of necessity eventually be subjected.

Presently, scintillation counter mapping of intrathoracic tumors following radioactive isotope administration is restricted to those tumors of suspected thyroid origin. As with thyroid tissue elsewhere, the uptake of I^{131} permits accurate identification by such technics. Insofar as is known, low millicurie dosages carry no short or long term hazard for the patient.

Some months ago I had the privilege of observing a demonstration of the new fluoroscopic image amplifier in action. With but momentary dark adaptation I watched, spellbound and incredulous, the movements of the vocal cords. The advantages of that equipment in the diagnosis of lung tumors I can only surmise, but it must be of very great help.

Mention should be made of the newer and safer bronchographic contrast media currently becoming popular. They are in aqueous solution, are rapidly and completely eliminated, largely by absorption, and the total examination period is shortened by their use. Shadows are not quite so distinct as with Lipiodol or Iodochlorol.

While body section roentgenography is certainly not new, it does now appear to be used more widely with respect to benign tumors. Its particular value is in demonstration of finer calcium deposits within circumscribed lesions when the deposits are not visible on conventional films.

Actually, today, exploratory thoracotomy is not the least reasonable solution to the diagnostic problem. The mortality rate is now very nearly that of exploratory laparotomy which physicians do not hesitate to recommend when the findings warrant it and when a definite diagnosis cannot be reached by more conservative means.

Obviously, the ideal treatment would be resection of the primary malignant lesions and those benign lesions which are considered hazardous. The other solitary circumscribed masses could be observed indefinitely. Unfortunately, accurate diagnosis is nearly always impossible by conservative methods. Until a better diagnostic approach is available, such lesions should be excised, since reports show that the resectability rate approaches 100 per cent in those solitary malignant lesions in which exploratory thoracotomy is performed before symptoms are produced.[2]

It is very important that an accurate pathologic diagnosis be made by frozen section at the time of thoracotomy. Simple excision, wedge resection, or segmental resection can then be used for the benign lesions and pneumonectomy reserved for the primary malignant ones.

Thoracotomy and empirical resection of all unidentified pulmonary masses are not suggested as a solution to the difficult diagnostic problem. Certainly each case must be considered on its own merits and as carefully studied as is practical without causing a possibly fatal delay before thoracotomy, which will be performed eventually in almost all cases.

I have endeavored in this discussion to review the present status of and recent advances in the diagnosis of benign pulmonary tumors. I have found the only reasonably new diagnostic method in wide use to be angiopneumography, and it is of limited benefit. Instead of new methods of roentgen diagnosis a new "roentgen attitude" has developed in which the benign pulmonary lesion becomes a practical problem with a logical therapeutic approach rather than an interesting academic entity without a constructive therapy.

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DISCUSSION

David M. Davidson, D.O., Muskegon, Mich.: The brief time allotted to the discussions on benign and malignant neoplasms of the lungs has produced excellent summarizations of present-day literature. Dr. Accola and Dr. Reed are to be complimented on covering such a volume of material in so short a time. The subjects that we have been concerned with in this discussion are:

1. The incidence of lung cancer
2. Danger signals
3. Need for follow-up examination
4. Treatment of nonoperable cases
5. Comparative accuracy of various examinations
6. Prognosis of benign and malignant lesions.

In summarizing the discussions I shall touch upon each of these topics in order, highlighting or reinforcing, for the most part, the points already made. In a few instances I shall call attention to conflicting opinions.

In my perusal of the literature, I find that chronic bronchitis is suggested by one author[1] as a conditioning factor in lung cancer. Some studies[2] have revealed that the lowest incidence of bronchogenic carcinoma

is found among agricultural workers and the highest is found in towns in which there are metal industries. As Dr. Reed pointed out, asbestos and metal fumes are already known to be agents. Another group[3] has noted that the highest incidence of pneumonia and lung cancer among laborers occurs mainly in those working out of doors. Death rates are higher for all occupational categories in areas of greatest air pollution than in cleaner areas. In both the cleaner and dirtier districts, lung cancer death rates of laborers are three times those of professional groups of the same areas.[4] Clemmesen[5] points out that a minimum exposure of 20 years is required.

Authorities vary considerably on what constitutes a danger signal. One group[6] has stated that in no case in which there was roentgenographic evidence of calcification in tumor was the lesion malignant. A second[7] cautions that, after the fortieth year, calcifications in a tumor should not be allowed to rule out the possibility of cancer. Pulmonary neoplasms in the presence of tuberculosis have been noted.[8-12] Pain syndromes which affect the extremities before pulmonary symptoms occur have been mentioned; these affect the joints mainly, are intermittent during the daytime, are independent of motion, and have no objective findings.[13] One study[14] led to the conclusion that no symptom was reliable or, in combination with others, was characteristic of malignant neoplasm.

In dealing with the patient, the general practitioner should place more emphasis on the importance of follow-up x-ray examinations. At the Muskegon Osteopathic Hospital there have been four cases of pneumonia which 1 year later were proved malignant. However, there was a reluctance on the part of all of these patients to return as requested for follow-up roentgenographic examination. It is felt that the roentgenologist should keep a record of any suspect case and inform the practitioner if this patient fails to return.

In the treatment of those malignant neoplasms which are not operable or for which surgery has been refused, adequate treatment will provide at least some relief from distressing symptoms. Endoscopic implantation of radon seeds in the more centrally located lesions is beneficial.[15] Radiation therapy will usually relieve the major distressing symptoms. It is believed by some[16] that nitrogen mustard is most effective in the presence of mediastinal obstruction and that injections of nitrogen mustard or radioactive colloidal gold into the pleural space may be helpful in pleural effusion.

The literature reveals a wide range in the percentage of accuracy given for each of the various procedures used to diagnose lung tumors. However, the consensus is that cytologic examination of sputum, bronchoscopy, biopsy, and secretion analysis are all valuable and should be done when possible.[17-22]

One additional source of information is the deep cervical fat pad and the contained lymph nodes.[23] If metastases are not evident and the aforementioned methods of diagnosis are inconclusive, exploratory thoracotomy should be performed with mediastinal and lung biopsy. Dr. Reed and Dr. Accola have noted that at present thoracotomy is little, if any, more dangerous than laparotomy.

The cinedensigraph is an additional method of tumor diagnosis.[24] Those familiar with this procedure found that in some cases it was the only method that indicated malignancy.[25] Cinedensigraphy is a photoelectric record of minute changes in amplitude density. Tracings of the lung and tumor are run simultaneously behind the fluoroscopic screen while electrocardiograms of the heart and greater vessel shadows are made.[25] Dr. Accola has pointed out the differences in the arterial and capillary formation in inflammatory, benign, and malignant lesions. It is these which account for the difference in tracings of the above lesions when reduced to the cinedensigram.

Dr. Accola also has noted that benign lesions have been taken from the realm of curiosity and placed on the list for active treatment. This change in attitude has been brought about by the improved methods of diagnosis and treatment. It has been noted that certain of these benign lesions have a tendency to degenerate. In alveolar cell tumor the structure is microscopically benign although the characteristics are those of a metastasizing lesion.[26]

In the prognosis of tumors, it has been noted that certain benign lesions have a tendency to degenerate into malignant forms while others of a benign form behave like metastatic neoplasms. It is doubtful whether any so-called benign lesion of the lungs should be given the opportunity to alter its character. Malignant lesions of central location metastasize early to the regional lymph nodes. Peripheral lesions show a later metastasis to the hilar regions. It must be further noted that the absence of lymph node involvement at thoracotomy is not an accurate guide to prognosis. It must be remembered that, when clinical manifestations warrant a tentative diagnosis, the probability of successful resection is already considerably less than in cases in which x-ray examination gives the first evidence of pathologic pulmonary findings.[27] The most important method of screening for pulmonary neoplasms is biannual posteroanterior and lateral teleroentgenography of all patients over the age of 50.

Third and Webster Sts.

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The Present Status and Recent Advances in the Radiologic Diagnosis of Gastric Ulcer

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During the past few years, the roentgenologic diagnosis of gastric ulcer has been undergoing a steady process of evolution. New developments have occurred not only in equipment and technic but, most important probably, also in diagnostic criteria. Concepts regarded as facts a few years ago are being modified or discarded, and other criteria are being added or substituted. A review of some of these changes is in order.

In the field of equipment, there have been several developments of importance. The filming fluoroscope or spot film device and the production of diagnostic tubes having smaller focal spots and larger heat capacities with accompanying transformers allowing high kilovoltage technics have been invaluable improvements. There is at least one commercial unit available for electronic intensification of the fluoroscopic image, permitting more accurate fluoroscopy and probably eventually more effective use of photofluorography. Roach, Sloane, and Morgan[1] of Johns Hopkins reported encouraging results in the experimental use of photofluorography as a screening procedure for gastrointestinal neoplasms. Further study of this technic seems warranted.

Along with changes in equipment have come improvements in contrast media. Chemists are providing more palatable media with smaller particles which stay in suspension better and provide a more uniform coating of the gastric mucosa.

Some changes in technic developed within the last few years are worthy of mention. Formerly, examination of the patient was delayed until the phase of gastric bleeding ended, thus causing a deferment in the application of proper treatment and, in the case of a small ulcer, giving the ulcer an opportunity to heal sufficiently to make its demonstration difficult or impossible. Now there is no fear of performing an examination during episodes of bleeding as long as palpation is restricted or eliminated and movement of the patient on the table is limited to a minimum. The rapid visceral survey technic of Gianturco[2] has made it more convenient for patients with limited time to present themselves for examination.

Certain developments in pharmacoradiography are of interest. These involve administration of morphine or insulin previous to re-examination of the stomach to differentiate areas of rigidity of the gastric wall due to spasm from those due to infiltration.

Roentgen technics for studying the digestion of fats and proteins have been developed using barium pills coated with fat or protein. These digestive nuclei, as they are called, are available commercially in Europe, but I know of no American source at this time. Serial observations are made following their ingestion to determine the digestive time of the fat or protein by their break-up in the digestive tract. The digestion time of both fats and proteins, but particularly proteins, is increased in patients with gastric hypoacidity or anacidity and decreased in patients with hyperacidity.

When confronted with a patient suspected of having a gastric ulcer, the roentgenologist has two problems: (1) he must find the ulcer if it is present, and (2) if he is fortunate enough to find the ulcer, he must try to determine if it is benign or malignant. There are, of course, no statistics available as to the number of ulcers that remain undiagnosed. However, some idea of the prevalence of gastric ulcer may be gained from autopsy statistics. Sturtevant and Shapiro found 2 per cent of autopsy cases in Bellevue Hospital showed either a gastric or duodenal ulcer; Welch, basing his figure on continental reports, estimated 5 per cent.[3] In 1952 Schindler[4] reported a series of gastric ulcers which were not demonstrable radiographically. Of seventy-four cases of proved benign gastric ulcers, visualized gastroscopically and examined by x-ray, 20 per cent were not shown on roentgenograms. The ulcers ranged in size up to 1.5 cm. in diameter. In this series, the most important factors in the x-ray failures seemed to be the shape and position of the stomach, with the high-positioned and steer-horn shaped stomachs giving the most difficulty. Statistics of Benedict and Tanner[5] show respectively 8 and 25.2 per cent of x-ray failures in a series of proved gastric ulcers.

The classical radiographic signs of gastric ulcer may be divided into two types, the direct and the indirect. There is only one direct sign, the demonstration of the ulcer niche, or crater, which is seen in profile as a protrusion of barium which may be conical, round, or mushroom-shaped. *En face*, the crater is round and may be surrounded by a halo of edematous gastric mucosa or by folds of mucosa radiating in a stellate pattern from the crater over the anterior and posterior walls of the stomach.

The indirect signs are as follows:

1. *Tenderness.*—This is usually described as well localized to the site of the ulcer with possible courses of radiation to the upper left abdominal quadrant or to the back. This sign, once considered important, is no longer relied upon so heavily because of its uncertainty.

Large ulcers may be demonstrated which show no palpatory tenderness. On the other hand, palpatory tenderness may be present in gallbladder, duodenal, or pancreatic disorders not related to gastric ulcer.

2. *Gastric Spasm.*—Gastric spasm is occasionally but not always an accompaniment of gastric ulcer. It is most commonly in the form of an indentation or incisura on the greater curvature opposite the crater. The incisura of the gastric ulcer must be differentiated from indentations due to the colon or spleen. The second most common form of gastric spasm is the antral narrowing occasionally associated with an ulcer of the lesser curvature. The narrowing is funnel-shaped and frequently persists after the ulcer is healed. Pylorospasm may also be present and may be transient or permanent; it may be severe enough to cause some dilatation of the stomach with increased gastric emptying time.

3. *Altered Peristalsis.*—The condition of the peristalsis depends on the site and duration of the ulcer. If there is no gastric obstruction, peristalsis is usually normal or increased. In the early stages of obstruction, peristalsis is increased in rate and amplitude. As obstruction progresses with dilatation of the stomach, peristalsis becomes decreased and eventually absent. Another peristaltic alteration of interest is Fränkel's sign or "peristaltic jump." This consists of an area of gastric wall at the site of an ulcer which shows no peristaltic action. The aperistaltic area may be large or small and is caused by local rigidity of the gastric wall due to scar tissue formation. In the presence of this sign, however, the presence of malignancy must be considered. This sign has not been found too practical, generally because it is so difficult to demonstrate. It was first demonstrated by cineroentgenography.

Carcinoma of the stomach is the most frequent cause of deaths due to cancer. In the United States in 1948, there were 26,215 deaths caused by cancer of the stomach; this constituted 12.6 per cent of all deaths from cancer.[6] Undoubtedly, then, the most important task of the roentgenologist is to differentiate benign from malignant gastric ulcers. Various criteria used from time to time to attempt the differentiation are as follows:

1. *Therapeutic Test.*—This has been advocated by many authorities who contend that rapid and complete healing of an ulcer under strict medical management is indicative of benignancy. Templeton[7] and Buckstein[8] both dispute this test and cite cases of ulcers that showed partial or complete healing with treatment, the apparent healing in some cases being due to ingrowth of carcinomatous tissue.

2. *Location of the Ulcer.*—Location has been considered by many authors as an important differential characteristic,[9,10] ulcers located on the greater curvature and the prepyloric portion of the antrum being considered malignant until proved otherwise. While it is true that many ulcers are malignant, benign ulcers are constantly being reported in these areas, with the ratio running as high as ten malignant ulcers to eight benign ulcers in a series of eighteen ulcers of the greater curvature reported by Silk, Blomquist, and Schindler.[11] The general incidence of benign ulcer of the greater curvature is estimated to be from 0.7 per cent to 14 per cent. On the other hand, ulcers of the lesser curvature, generally considered a benign area, have a 10 per cent possibility of being malignant, while as many as 20 per cent of those of the anterior and posterior walls may be malignant.

3. *Size of the Crater.*—Size has long been used as a guide to whether an ulcer is malignant or benign. Generally, an ulcer 2 cm. or less in diameter is considered benign, while one 2.5 cm. or more in diameter is considered malignant. This guide is now less generally used than it was a few years ago. Templeton[7] reported benign ulcers measuring up to 15 cm. in diameter and ulcerating carcinomas having craters of 2 cm. or less.

4. *Characteristics.*—Various characteristics of the crater and surrounding mucosa are often cited as denoting benignancy or malignancy. Thus, small conical craters arising in the lesser curvature with little or no surrounding edematous cuff are generally regarded as benign as are those larger ulcers that show overhanging edges, protrude beyond the gastric wall, and/or show the presence of a fluid level topped by gas. Mucosal folds radiating from the crater in a stellate pattern are also believed to be indicative of a benign ulcer.

Rigidity of the gastric wall on either side of the crater is taken as an indication of malignant infiltration, although it may also be caused by fibrosis. A typical malignant crater is irregular in outline, not projecting beyond the gastric wall, and shows a loss of normal rugae surrounding the ulcer. An ulcer of this type, particularly if demonstrated as arising within an intragastric mass, poses no problem in diagnosis. The difficulty in diagnosis arises when a benign ulcer has the characteristics described above. Practically all of these characteristics have been refuted by one or more authors. To give one illustration, Cole[12] stated that the best example of stellate mucosal folds he had seen prior to 1933 was in a malignant ulcer of the lesser curvature arising in a carcinoma, the bulk of which extended outside the stomach.

Thus, it can be seen that, with the accent on earlier diagnosis of cancer and the propensity of early malignant ulcers to imitate benign ones, roentgenologists have an obligation to use every means at their disposal to eliminate the possibility of malignancy whenever an ulcer is found and to consider each ulcer malignant until it is proved otherwise. This sentiment was forcefully expressed by Weber[13] of Mayo Clinic when he said:

> Radiologists can make a significant contribution to the diagnosis of early, that is curable, gastric cancer, if at the time of their examination, they will temporarily refuse to recognize such a thing as a benign gastric lesion. In my opinion it is difficult to justify the use of any word in our reports that has any connotation, however remote, of benignancy. This holds for the gastric ulcer regardless of its size or other physical features and it holds for the gastric polyp as well.

While it is true that the incidence of malignancy in these lesions is only 5 per cent to 15 per cent, when it comes to the evaluation of each individual case, roentgenologists cannot afford to gamble with probabilities. They must insist upon the opportunity to re-examine patients in order to obtain adequate mucosal studies, secure proper interval examinations to check on the healing of the lesion, and make free use when possible of laboratory, gastroscopic, cytologic, and clinical aids to insure adequate diagnosis.

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Polyps of the Colon

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Many different types of lesions must be considered in discussing polyps of the colon. The term "polyp" refers to any pedunculated growth from a mucous surface. Included in this classification are lesions such as simple epithelial hyperplasia, adenoma, lipoma, adenomyoma, fibroma, angioma, lymphoma, and many others. The adenomatous type of polypoid lesion constitutes the most common benign neoplasm of the colon.

A large percentage of carcinomas found in the colon and rectum have had their origin in a benign polyp. Ackerman and Regato[1] state that the presence of polyps is the most important single etiologic factor in carcinoma of the large bowel. It is also an accepted fact that the hereditary or familial type of polyposis must be treated radically because the condition invariably results in malignant transformation. In consideration of these facts, the importance of early recognition and appropriate treatment becomes apparent.

INCIDENCE

Contrary to the popular belief that benign tumors of the bowel are relatively rare, these lesions are being found with increasing frequency as better methods of examination are developed. Dukes[2] reports one or more simple tumors at some point in the bowel between the cecum and the sigmoid in 9.4 per cent of patients dying from causes other than carcinoma of the bowel.

Most important of the inflammatory conditions are chronic ulcerative colitis and intestinal parasites. The greatest portion of the large bowel is constantly being subjected to irritation by the passage of its normal contents. That something besides irritation and inflammation is necessary for the development of polypoid growth is evidenced by the fact that so many escape this affliction. The theory has been advanced that the normal mechanism governing orderly growth is an inhibitory one and that tumor formation is not the result of stimulation of growth per se, but is due to a disturbance in the inhibitory regulation.[3]

It has been fairly well proved that in some cases of multiple polypoid lesions of the bowel there is a familial disposition or hereditary character.

Embryonal rests in the wall of the bowel have been recorded, as they have been in other structures of the body. Teratomas, dermoid cysts, and enterogenous cysts are, without question, embryonic in origin.

Most tumors of the intestine occur in the large bowel, and they increase in number from the proximal to the most distal portion of the colon. More than 66 per cent of the intestinal tumors occur in the colon, and of these about 70 per cent are found in the rectum.[3]

APPEARANCE

Every variety of polypoid lesion, from small excrescences to large pedunculated masses, may be found. Lesions may be single or multiple and diffuse, involving practically the entire bowel surface. When multiple, all varieties in different stages of development may be found in the same colon. The lesions may vary in size from a few millimeters to 10 cm. or more in diameter. The average size varies from 2 mm. to 2 cm. The color varies from a pale pink to a dusky red, and, as a rule, the larger the lesion the darker the color.[3]

The structure of polyps varies markedly from localized epithelial hyperplasia to a mass which is indistinguishable from adenocarcinoma. As the tumor grows and develops, changes take place, particularly in the periphery of the lesion, and eventually a malignant status is reached.

CLINICAL FEATURES

Polyps may be present for many years without producing any symptoms. In many cases they are discovered incidentally on routine investigation. Symptoms, when present, depend on the size and location of the lesions, the number of lesions, the extent of colonic involvement, and the presence or absence of complications.

Age.—Polyps are most commonly found in persons of advanced middle age or older, although they have been encountered in children as young as 6 months of age.

Sex.—Males are affected more often than females in a ratio of three to two.[3]

Race.—Polyps are more common in persons of the white race than in those of the Negro race.

Symptoms.—Symptoms include blood in the stool, which occurs in about two-thirds of cases;[3] diarrhea, which is present in a smaller percentage of cases; abdominal pain in approximately 25 per cent of cases; and painful or difficult defecation. Occasionally a polyp may protrude from the anus, and, very rarely, a polyp

may be pulled loose from its narrow stalk and be spontaneously expelled.

Clinical Course.—The symptoms may persist unchanged for many years or throughout the life of the patient unless complications take place. In the hereditary or familial type, complications are usually inevitable, and the clinical course is progressively downward.

Laboratory Findings.—The laboratory is of little help in establishing a diagnosis. There may be mild anemia, and some degree of leukocytosis is to be expected in complicated cases with infection. The sedimentation rate will vary with the degree of tissue destruction, and occult blood will be found at some time or other in the stools in two out of three cases.[3]

EXAMINATION

Most benign growths of the colon occur in the region which can be explored by the sigmoidoscope. From two thirds to three fourths of all polypoid lesions can be seen through the standard 25-cm. scope.

Sigmoidoscopy is considered essential to the study of all patients with symptoms and signs of colonic dysfunction or disease. With proper training and equipment, it is a relatively simple procedure which requires no anesthetic and can be carried out without an elaborate operating room setup. By observing a few simple rules the examination is comparatively painless and without danger of serious accident or complications.

The following rules should be observed:

1. Digital examination of the rectum should be performed before the scope is passed.
2. A small caliber instrument, 1.5 cm., should be employed.
3. The instrument should never be blindly inserted further than the finger has explored.
4. The instrument must never be forced against the bowel wall until the lumen ahead is identified.
5. Frequent air inflation should be used.
6. The instrument must never be forced past a strictured area or an area where undue resistance is met.
7. The instrument should never be passed while the patient is under general anesthesia.
8. The patient should be in the inverted position. The knee-chest position is not satisfactory for this procedure, and many bowel perforations have resulted from its use. In the extremely weak, aged, or cardiovascular patient, the left lateral Sims' position should be substituted for the inverted position.

In patients with diarrhea or bleeding, no preparation should be made in order that the membranes can be studied without having been subjected to preliminary trauma. If enemas are necessary, plain water or isotonic salt solution should be used. The examination can be carried out 2 to 4 hours after the injection.

ROENTGEN EXAMINATION

Roentgenoscopy is the only satisfactory method of demonstrating lesions in the colon above the sigmoidoscopic level. This examination is essential in the investigation of any patient with colonic symptoms or in anyone where there is suspicion of colonic disease.

The following roentgen procedures are available, and each has its advantages and disadvantages:

The Preliminary Abdominal Survey Film.—This survey film is routinely taken in the x-ray department at the Fort Worth Osteopathic Hospital, and many times provides the key to diagnosis.

Oral Barium or the Progress Meal.—This procedure is of some value in routine investigation of the colon but is not essential and many times is contraindicated. Oral barium, of course, should not be administered if there is any indication of bowel obstruction.

Barium Enema.—The opaque enema is essential in nearly all colon problems and has many advantages over the oral mixture.

1. It is possible to fill the entire colon, unless obstruction is present, and to study it in all positions by roentgenoscopy.
2. It is less time consuming and less expensive.
3. There are few contraindications to its use.

Barium-Air Double Contrast Enema.—It is generally agreed that this method is most valuable in demonstrating polyps, small carcinomas, and ulcerative lesions. Immediately following exposure of the film taken after the evacuation of barium, the colon is insufflated with air under fluoroscopic control. The air is introduced slowly, and care is taken against overdistention. When the colon is filled, films are exposed in prone and lateral or oblique positions as indicated. Polypoid tumors are visualized as projections into the intestinal lumen which stand out in sharp contrast in the air-distended bowel. Suspicious lesions should be re-examined and proved to be constant before radical treatment is advised. According to Weber,[4] the following roentgen signs are diagnostic of a benign tumor in the colon:

1. Smooth, sharp, evenly rounded, air-bubblelike filling defects (These often may be pushed up and down for a few centimeters and are best brought out by manually compressing the bowel walls.)
2. No change in the outline of the colon itself
3. Freedom from involvement of the colonic musculature
4. Pliancy of the bowel walls
5. Absence, with rare exceptions, of any associated palpable mass.

Thorium-Air Double Contrast Enema.—The basic advantage of this method is that thorium preparations are precipitated or flocculated in water and that a flocculent medium will adhere to the mucosal surface much better than ordinary barium. There are three preparations which are rapidly precipitated in water and are considered satisfactory. They are: Collothor, Umbrathor, and Diagnothorine. This method is time consuming, expensive, and, unless the technic is followed very carefully, deposition of the opaque medium on the mucosa may not be successful. The technic is as follows:

1. The colon must be completely free from fecal material and mucus.
2. Since proper precipitation of the thorium depends on the hydrogen ion concentration of the bowel content, a solution of sodium phosphate, 10 to 20 per 1,000, must be used as a colonic lavage 1 hour prior to injection of thorium.
3. The thorium solution must be diluted in distilled water and given very slowly so as to fill the colon without distending it.
4. Films are exposed, and the patient must remain quiet for 15 minutes for flocculation to take place.
5. Evacuation is then permitted but straining is prohibited, following which further films are exposed.
6. Following this the colon is filled with air, and additional films are exposed.

DIAGNOSIS

If every person who complains of symptoms referable to the colon had the advantage of a thorough sigmoidoscopic and roentgen examination, few cases

of polyposis would be overlooked. These are the two methods by which a large percentage of these lesions can be diagnosed, and they are used far too infrequently. The examination must not stop with the discovery of a polyp or multiple polyps in a given area. The entire colon must be examined to determine the extent of dissemination. Specimens must be taken for biopsy and numerous sections examined for evidence of malignancy. And last, but very important, other members of the immediate family should undergo similar examinations.

TREATMENT

The treatment of polyposis can be summed up in a few words.

1. There is no medical cure.
2. X-ray treatment can be expected to give symptomatic relief, but cure is uncertain.
3. Some type of surgical management varying from simple fulguration to total colectomy with permanent ileostomy will be the best method of treatment in most cases.

CONCLUSIONS

Colonic adenomas are not uncommon lesions, but neither are they innocuous. Their early detection and proper treatment are imperative if the incidence of carcinoma of the colon and rectum is to be reduced.

A careful case history, which should include a family history, is probably the most important part of the examination. In all patients whose history is suggestive of colonic disease or dysfunction, a thorough sigmoidoscopic and roentgenologic examination is essential. The future health of these patients depends entirely on the ability of the sigmoidoscopist, the roentgenologist, and the surgeon to detect and properly treat these potentially malignant lesions.

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Roentgen Therapy of Some Common Degenerative Lesions

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The problem of malignancy, its diagnosis, and its management has assumed such a prominent place in lay and professional thinking during the last decade that a tendency to place a lesser importance on benign lesions has developed. This seems to be especially true among therapeutic radiologists, who have at their command a sometimes remarkably effective modality for the management of simple degenerative problems, but who tend to disregard or minimize its application in nonmalignant diseases.

This is unfortunate for both the patient and the radiologist. Much relief may be granted to a patient afflicted with a chronic or acute degenerative problem which threatens neither life nor limb, but which does result in a lesser or greater degree of disability and discomfort. The radiologist who encounters these situations in the diagnostic service which he renders must take the lead in thinking of therapeutic radiation. He cannot expect that the average nonradiologist physician will be the leader in this regard. Much as a physician immediately turns to radiation in certain malignant diseases, he will turn to radiation only as a last resort in many nonmalignant, so-called simple degenerative and inflammatory entities.

These contentions seem to be at variance with the opinions of some radiologists, who argue that benign lesions, especially the more common degenerative and inflammatory ones, should not be treated with a modality which can produce lasting tissue change and systemic effects. It is undoubtedly well to approach radiation therapy with caution, and it is wise to respect the potentialities of the weapon which one is wielding. However, the man, not his tool, must remain the master. Properly applied, radiation can be more effective and, possibly, less harmful than many other modalities which are utilized in such diseases.

The entities to be considered are the common inflammatory and degenerative articular, periarticular, and soft tissue lesions such as degenerative joint disease, acute and chronic synovitis, peritendinitis, both calcified and noncalcified, bursitis, discogenic spondylitis, idiopathic radicular syndromes, capsulitis, and the scapulocostal syndrome.

The purpose of radiation in such situations is essentially twofold. One aim is to relieve pain and disability; the other is to alter the progress of the degenerative or inflammatory process. The relief of pain is often easily accomplished and in most cases is readily assessable. The alteration of the basic pathologic process is probably less readily accomplished and is certainly less easily analyzed.

The lesions which were enumerated all possess an inflammatory and a degenerative component which vary in their combinations. The relative efficacy of radiation often depends upon the predominence of the inflammatory over the degenerative changes. Degenerative joint disease of a knee or a finger, being a more or less local mechanical structural problem without any known significant systemic or focal physicochemical change, will be affected less dramatically than synovitis or tendinitis, which possesses a distinct physicochemical pathologic physiology and may even bear a relationship to systemic changes.

Not a great deal is known concerning the physiologic chemistry of periarticular connective tissue. Since the fundamental action of radiation is to produce physicochemical alteration of tissues by means of ionization, the precise mechanism of effect in these disease entities cannot be postulated. However, certain facts suggest that radiation in these situations is a rational approach. Connective tissue consists of fibers and interfibrillar cement substance. The fibrous ele-

ments are either collagenous, reticular, or elastic fibers and basically are chains of protein molecules of high molecular weight. That the collagenous elements are chemically complex, and that they are intimately related to systemic body chemistry, has been repeatedly demonstrated in recent years. It has also been realized that certain collagen diseases are responsive to radiation therapy.

The cement substances are also composed of complex protein molecules but possess mucopolysaccharides as part of their structure. Among these mucopolysaccharides is hyaluronic acid, and it seems that those connective tissue elements containing high concentrations of hyaluronic acid may respond to radiation better than those containing lesser concentrations. As an example, the synovial fluid contains only hyaluronate, while cartilage contains none. In acute joint effusions, which often respond well to radiation, the concentration of hyaluronic acid is abnormally high. Thus, in addition to the classical elements of inflammation such as hyperemia and cellular invasion, these articular and periarticular lesions probably present chemical changes peculiar to their special physicochemistry which may be altered by radiation.

Although the degenerative components usually are considered irreversible as far as radiation is concerned, it seems to me that as long as progressive changes are occurring there is some foundation for belief that the process may be altered by roentgen therapy. Young fibroblasts are radiosensitive cells, and if they can be destroyed or if their activity can be modified, it is possible that some of the fibrosis may be prevented, or at least that its development may be restrained sufficiently to alter the end-result of the process.

Radiation therapy of these lesions begins with an adequate history and physical examination. Traumatic lesions and neoplasms, specific infections, and metabolic problems may masquerade as acute and chronic synovitis, peritendinitis, and the like. A preliminary roentgen examination of the involved area is indispensable, both for the purpose of ruling out these possibilities and for evaluating proper therapeutic approaches. If there is any doubt concerning the diagnosis, it is wise to withhold therapy until further clarification is obtained.

The roentgen technics which I employ in the management of these lesions fall into two or three basic patterns, depending upon whether the process is acute or chronic and upon the area involved. An episode is considered acute if it is of sudden onset and of short duration, perhaps no more than 72 hours; if it is severely painful and there is marked limitation of motion; and if it is accompanied by evidence of inflammation such as erythema, heat, edema, and pressure tenderness. Not all these criteria are necessarily present in every acute episode, and the acute radiculitis and paraspinal problem does not present surface evidence of local inflammation. The lesion is considered to be chronic if its duration is longer, if it is not as severely symptomatic, if motion restriction has been present for more than a few days, and if there is visual or palpatory evidence of soft tissue atrophy or fibrosis. Occasionally soft tissue calcifications, if extensive, can be palpated beneath the skin in bursal or tendinous areas, in both the clinically acute and chronic cases. Pressure tenderness may be observed in acute and chronic syndromes, although it is usually well localized in a chronic problem and more extensive in an acute episode. Often, in acute peritendinitis, synovitis, or radiculitis, a history of trauma or of a sudden, severe traumatizing movement may be elicited, suggesting the possibility of a true linear fracture or of an avulsion mechanism.

Although these clinical criteria apply most specifically to peritendinitis in an extremity, they may be modified, allowing for anatomic and functional factors, for the peripheral joint and vertebral and paravertebral inflammatory and degenerative situations. Thus, acute synovitis, such as often occurs in the knee joint, may be severely painful, motion may be extremely limited, the joint will be tender to pressure, and visual and palpatory evidence of edema will be present. A history of trauma immediately preceding the onset of symptoms is common. Chronic, recurrent synovitis is usually not tender, but it is painful with prolonged weight bearing or with certain movements. Radiculitis, whether of cervical, dorsal, or lumbar origin, having degenerative disease as its etiologic factor, or apparently idiopathic in nature, can be approached with similar fundamental criteria. There may be an extremely sudden onset of severe, lancinating radicular pain, with marked motion restriction and spasm in the spinal area involved, weakness of the involved extremity or extremities, and paresthesias. The nonacute case may present only recurrent pain or recurrent paresthesias in the absence of pain, no restriction of vertebral motion or acute spasm, and some exaggeration of the symptoms with certain vertebral column motions. Acutely afflicted patients may show marked tenderness to pressure over the entire nerve area, while localized tender segments sometimes can be detected in those with chronic afflictions. Occasionally slight atrophy of an extremity is associated with a protracted process while edema of an extremity can accompany the acute syndrome.

There are two other common inflammatory lesions of the same general type which often respond well to roentgen therapy. One is fairly well defined, in both clinical and pathologic aspects, as the scapulocostal syndrome. This is supposedly an inflammatory involvement of the bursa, which lies beneath the supermedial angle of the scapula. It is manifested by pain in the cervical area, shoulder, and upper extremity on the affected side, and by pain directly over the supermedial angle of the scapula, which is exaggerated by pronation of the shoulders and somewhat relieved by extension. The acute case exhibits exquisite tenderness over the bursa, and the pressure will reproduce or exaggerate the pain in the neck, shoulder, arm, forearm, or hand.

The second of the additional syndromes is less easily described and classified. If it occurs in the dorsal area, it consists of localized pain and localized pressure tenderness over the heads of one or more ribs; if it occurs in the lumbar region, the pain and tenderness occur over the tips of one or more transverse processes. For lack of a better term, this might be labeled "capsulitis" when it is located in the region of the costovertebral articulations. Some patients with these findings, who have responded to x-ray therapy, had failed to respond satisfactorily to manipulative management.

Another group of degenerative, inflammatory lesions is composed of the common degenerative joint disease processes involving the vertebral column and the peripheral joints, especially the knees and hands. Clinically and pathologically the degenerative element predominates over the inflammatory, and probably it is the presence or absence of associated synovitis, soft tissue

edema, and other local secondary inflammatory changes which determine the response to roentgen therapy.

There are many accepted approaches to the roentgen therapy of such a group of entities. Perusal of the literature will reveal a wide range of total dosage, physical factors, and degree of protraction. Roentgen therapy of these disabilities becomes, therefore, more or less a personal choice, depending upon how one evaluates the pathologic state and rationalizes the modus operandi of the radiation. The technics to be outlined now are therefore my approach to the problem and are not meant to be the generally accepted or the only permissible procedures. Nor are these technics new in the sense of presenting heretofore unappreciated ideas. They simply represent what I consider to be a workable, usable formula, developed as a result of my formal radiologic training and of treating many of these lesions in the past few years.

Fundamentally, the therapeutic approach is that which is applicable to benign, inflammatory processes, including limitation of total dosage to not more than the threshold erythema dose; fractionation over a period of 12 to 20 days in an acute problem, or longer in a markedly chronic lesion; gradually decreasing port size as the symptomatology is controlled, if such is possible; increased intervals between treatments toward the end of the course of therapy as compared to the early treatments; and in the chronic problems, gradually diminishing dosage at successive treatments. This basic pattern is applicable to all the entities previously discussed, with variation depending upon the acuteness or chronicity of the symptoms.

The physical factors nearly always employed are 200 kv., at 50 cm. distance, utilizing a half-value layer of 1.0 mm. Cu. Occasionally, if the area to be treated is very superficial and the part thin, 140 kv. at 36.5 cm. distance, with an HVL of 0.4 mm. Cu., is substituted.

Acute peritendinitis, the scapulocostal syndrome, capsulitis, radiculitis, or synovitis is given an initial dose of 150 roentgens in air with the 200-kv. technic through a portal which is large compared to the area of involvement. Thus, the initial shoulder portal size may be 15 sq. cm. and the initial port for elbow or wrist tendinitis 10 sq. cm. A 15-sq. cm. cone, triangulated, to cover some of the trapezius muscle on each side, is usually employed for the initial treatment to the cervicodorsal area; 150 roentgens are again delivered through an appropriate second portal, if available, in 3 or 4 days, depending on the acuteness of the symptoms at the initial visit. If the symptoms are subsiding satisfactorily, the interval is extended to 5 days, at which time 150 roentgens are delivered through a third port, if possible. The fourth and last treatment is again at a 5-day interval, and 100 to 150 roentgens in air are administered through an available portal. The portal size is reduced at some time during the course of treatment if the symptoms are diminishing in severity and are localizing in extent, although it is never reduced to less than 10 sq. cm. for a shoulder, 10 by 15 cm. for a cervical spine, or 6 sq. cm. for an elbow, wrist, foot, et cetera. Thus, an acute case receives 550 to 600 roentgens in air within 10 to 15 days. If the lower kilovoltage technic is employed, the dosages are limited to 100 roentgens per treatment, at the same fractionation.

The major portion of the acute cases will have greatly improved by the completion of this cycle. However, regardless of the symptoms remaining, treatment is discontinued when 600 roentgens have been delivered, and the patient is evaluated at the end of 2 weeks. Most patients show additional improvement during this period, and occasionally, improvement is continuous up to 1 month following completion of treatment, which probably is the minimum interval which should elapse before retreatment is considered. It is interesting to speculate that there may exist a biologic basis for this clinical observation, since 28 days classically represents the end-point of the main erythema phase of a skin reaction. After the 1-month interval, or after a still longer period if it was deemed desirable to wait, if symptoms persist and retreatment is decided upon, the lesion may then be considered to be chronic.

The occasional case of acute peritendinitis with overlying edema and erythema can often be adequately managed with diminishing doses given at 24- to 48-hour intervals, with a total of perhaps 350 to 400 roentgens in air in three or four treatments. The choice of this technic or of the technic of administering 150 roentgens four times, or of an intermediate method depends upon the clinical impression at the initial treatment.

Chronic states require a somewhat more prolonged treatment course, although the dosage per cycle is still limited by the total erythema dose of the technical factors being employed.

In the average shoulder case with low grade symptoms, the total of 600 roentgens in air is delivered in fractions of 150 roentgens each at 5-day intervals, different portals being employed each time. The 2- to 4-week waiting and observation period is more important in these cases than in the acute cases, since the response to treatment is almost invariably a slow, gradual one which takes some time to manifest itself. Usually no effect is noted until about 12 to 15 days following the initial treatment, that is, until about the third visit. Occasionally, improvement does not begin until the last visit, or even until some time during the post-treatment observation period.

The same technic is applied to other areas of chronic tendinitis, such as the wrist area, elbow, trochanteric region, knee, ankle and tarsal areas, as well as to chronic degenerative spondylosis anywhere in the spine, and to the capsulitic and scapulocostal syndromes.

Occasionally, an interval of 7 days is allowed between the third and fourth treatments, spreading the 600 roentgens over a 22-day period. This is done in cases showing a somewhat slower than average response.

The portal size is usually kept large throughout the course of treatment in chronic cases, the reasoning being that the musculofascial changes which accompany and result from a long-standing pathologic condition are often extensive as compared to the actual site of the initial lesion. As an example, a 20 by 20 cm. port might be used posteriorly in a chronic shoulder problem, in an attempt to produce some effect in the trapezius region, which is often tender and spastic.

Even greater protraction and fractionation are sometimes used in chronic tendinitis of the distal extremities and in indolent synovitis of the knee and ankle. Such lesions, if of long standing, and especially if they are constantly aggravated by weight bearing and occupational necessities, often can be coaxed into response by small doses of 75 to 100 roentgens over a period of several weeks. The total dose should not ex-

ceed 600 roentgens. The first two or three applications may be at 5-day intervals, with the remaining at weekly intervals, until response is being manifested or until 600 roentgens have been given.

The consideration of a second course of treatment is never entertained until the 4-week interval examination and is rarely instituted until 8 to 10 weeks following completion of the initial cycle. The indication for retreatment is persistence of the patient's symptoms, provided that there has been evidence of at least some radiation response during the first treatment course. If no response to the first cycle was evinced—if there was no change in the distribution, character, or intensity of pain or paresthesias, if the range of motion has not increased, even minimally, if edema is still present and is unchanged—no further radiation is administered. If some change in the presenting syndrome has occurred, and if it is felt that the response has run its course but that additional treatment might produce further improvement, a second course of treatment is undertaken. This general plan is based upon the impression that if a syndrome is susceptible to radiation, it will exhibit some response to the initial course, and that retreatment in instances of no detectable response to the first series is of no value.

When retreating a lesion, it should be managed as a chronic problem, regardless of the clinical state at the start of the initial course.

The only adjunctive therapeutic measures employed are rest of the part in all acute syndromes, and exercises designed to encourage stretching of the soft tissues in those chronic lesions which are accompanied by fibrosis and contraction. Other forms of physiotherapy are prohibited during roentgen treatment. Patients with a scapulocostal syndrome are urged to extend the shoulder and neck as persistently as possible. Osteopathic manipulative therapy to the cervical area in the degenerative spondyloses with radicular pain seems to be valuable in conjunction with radiation.

This has been a brief review of personal experiences with a few common inflammatory and degenerative problems. Contrary to some opinions, I am convinced that roentgen therapy has a definite place in their management, provided that proper and careful individualization of treatment is provided.

59 E. 54th St.

BOOK NOTICES

(Continued from page 754)

CLINICAL INTERPRETATION OF LABORATORY TESTS. By Raymond H. Goodale, M.D., Pathologist at the Worcester Hahnemann Hospital, Fairlawn Hospital, and Worcester County Sanatorium, Worcester, Mass., Clinton Hospital, Clinton, Mass., Harrington Memorial Hospital, Southbridge, Mass.; Consulting Pathologist, Veterans Administration Hospital, Rutland Heights, Mass. Ed. 3. Cloth. Pp. 720, with illustrations. Price $7.50. F. A. Davis Company, 1914-16 Cherry Street, Philadelphia 3, 1954.

This is the third edition of this work and contains considerable new material, added because of advances in the field since the previous edition. It is principally a brief, comprehensive cataloguing of the highlights of laboratory results as applied to diagnosis. The brevity of presentation and lack of descriptive detail should facilitate its use as a quick reference, but they also make the collateral use of other texts unavoidable in most cases. Unfortunately, the index is not as complete as it could be for optimum usefulness.

SHOULD THE PATIENT KNOW THE TRUTH? A Response of Physicians, Nurses, Clergymen, and Lawyers. Edited by Samuel Standard, M.D., and Helmuth Nathan, M.D. Cloth. Pp. 160. Price $3.00 (hard cover), $2.00 (soft cover). Springer Publishing Company, 44 E. 23rd St., New York 10, 1955.

It is not certain for whom this book was written. It contains little that the physician has not already figured out for himself and is somewhat technical for lay readers. However, there is considerable material of interest, and it will probably console the physician whose conscience is hurting him. The conclusions that are drawn are just about what you would expect—it all depends on the patient, the case, the time, and the circumstances.

HEMATOLOGY. By Cyrus C. Sturgis, M.D., Professor of Internal Medicine, Chairman of the Department of Internal Medicine, University of Michigan Medical School; and Director of the Thomas Henry Simpson Memorial Institute for Medical Research, University of Michigan, Ann Arbor, Michigan. Ed. 2. Cloth. Pp. 1222, with illustrations. Price $19.50. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1955.

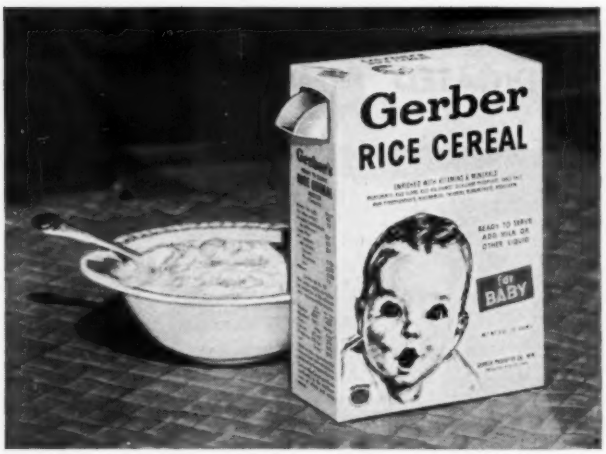
This is a revision of the original text which was published in 1948. Much new material has been included because of the advances in hematology since the previous publication. The material is complete and well organized, with an abundance of detail; in many cases the physician will find more than he wants to know. Sections on treatment are sufficiently detailed for practical use, and the sections on diagnosis are extremely well developed. There are nine color plates, well done but not separately indexed. This work is in no sense an atlas of hematology for use in the laboratory, but rather is directed at the actual handling of cases of hematologic disorders. It should prove to be most useful.

ANCIENT THERAPEUTIC ARTS. The Fitzpatrick Lectures delivered in 1950 & 1951 at the Royal College of Physicians. By William Brockbank, M.A., M.D. (Camb.), F.R.C.P. Cloth. Pp. 162, with illustrations. Price $5.00. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1955.

This is an interesting and well-written description of the chief of the ancient therapeutic arts. It has no practical value to the physician of today, but it will add to his knowledge of medicine of a former day and make him glad that he was not a patient in that era. Among the methods described are clysters, cupping, and setons. There is also a fascinating section, Intravenous Injection of Drugs. It was a matter of interest and surprise, to this reviewer at least, that intravenous fluids were used fairly extensively and with some success in cholera in the early nineteenth century!

HANDBOOK OF TREATMENT. By Harold Thomas Hyman, M.D., author of Integrated Practice of Medicine and Handbook of Differential Diagnosis. Cloth. Pp. 511. Price $8.00. J. B. Lippincott Company, East Washington Square, Philadelphia 5, 1954.

This book is just what the name implies. The entire emphasis is on therapy, and the format of the book is that of a quick reference source rather than that of an expository work. Diagnosis is included only insofar as it is pertinent to the treatment at hand; the author assumes that a diagnosis has already been made. The work is indexed by disease and by drugs, the latter by both general group and specific entries. Exact dosage schedules are given only when essential to the use of the drug. In the disease sections, general principles of management are included as well as specific therapy. This is not a work that can be used with any great degree of success without fuller references available, but it should prove to be a great time saver in many cases.



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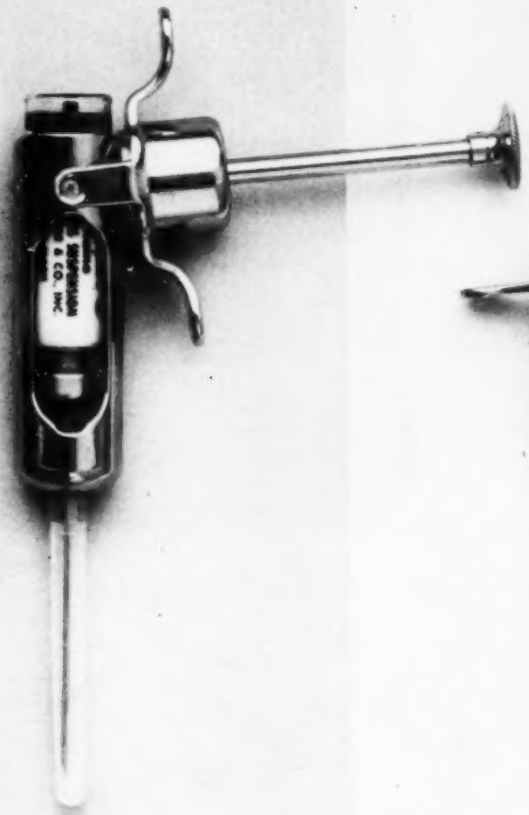

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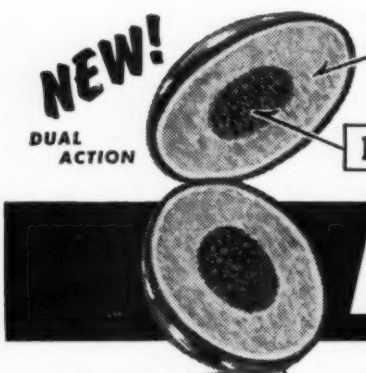

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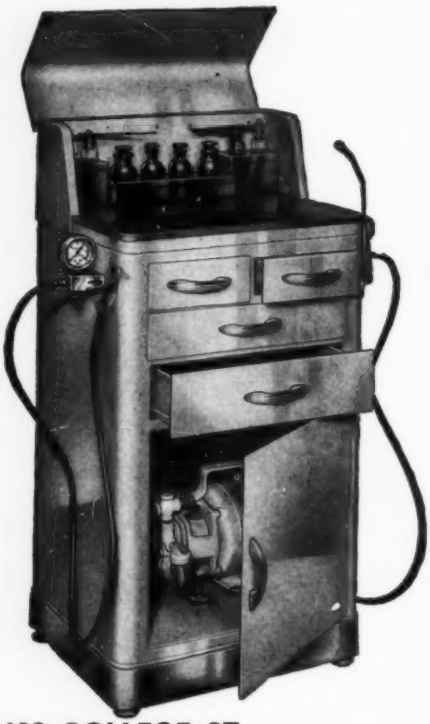

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RIASOL'S unique formula has met with outstanding success in psoriasis. In most cases the unsightly lesions yield promptly to its effective action and often do not recur for long periods. Itching, if present, is usually alleviated.

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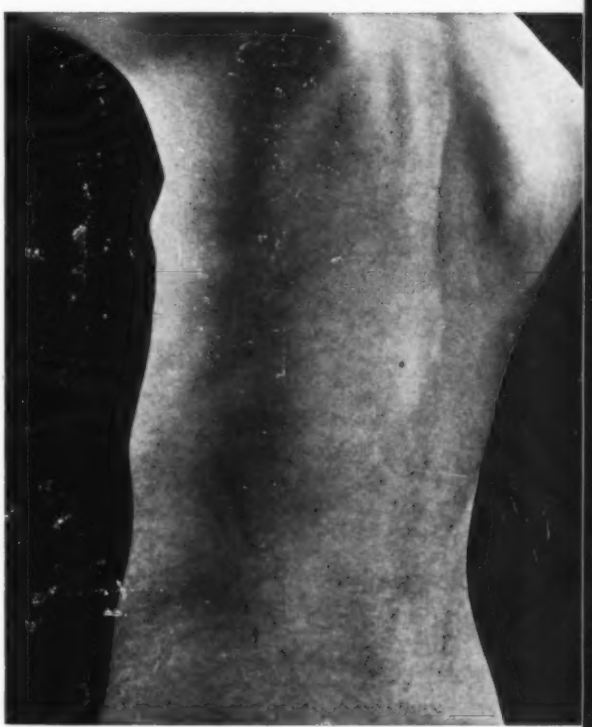

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Conventions and Meetings

Announcements

American Osteopathic Association, Sixtieth Annual Convention, New York City, July, 1956. Program Chairman, Myron C. Beal, 17 Vick Park A., Rochester 7, N.Y.

American College of Osteopathic Internists, annual meeting, Sheraton-Cadillac Hotel, Detroit, October 20-22. Program Chairman, Ward E. Perrin, Chicago Osteopathic Hospital, 5250 S. Ellis Ave., Chicago 15. Secretary, Glennard E. Lahrson, 460 Staten Ave., Oakland 10, Calif.

American College of Osteopathic Obstetricians and Gynecologists, annual meeting, Neil House Hotel, Columbus, Ohio, February 6-9. Program Chairman, Richard E. Eby, 1247 N. Park Ave., Pomona, Calif. Secretary, Harold K. Morgan, 3268 W. 32nd Ave., Denver 11.

American College of Osteopathic Surgeons, annual meeting, Hotel Statler, Washington, D.C., October 30-November 3. Program Chairman, Arthur M. Flack, 135 S. 17th St., Philadelphia 3. Executive Secretary, Orel F. Martin, Box 474, Coral Gables 34, Fla.

American Osteopathic Academy of Orthopedics, annual meeting, Hotel Statler, Washington, D.C., October 29-November 2. Program Chairman, Karl P. B. Madsen, 460 Staten Ave., Oakland 10, Calif. Secretary, J. Paul Leonard, 2673 W. Grand Blvd., Detroit 8.

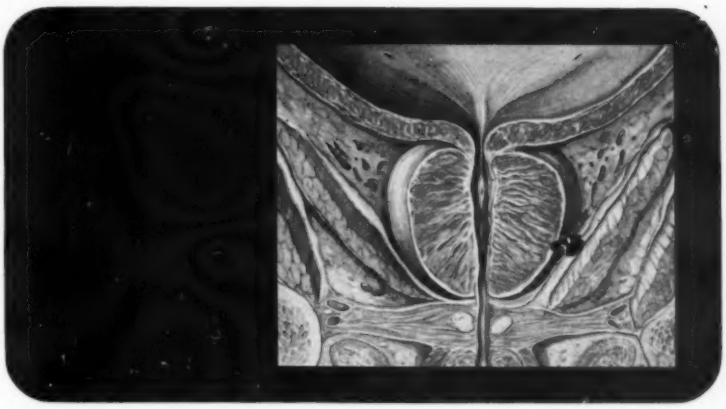
American Osteopathic College of Anesthesiologists, annual meeting, Hotel Statler, Washington, D.C., October 30-November 3. Program Chairman, Harry J. Petri, Jr., 5 Deering St., Portland 3, Maine. Secretary, Crawford M. Esterline, Box 155, Kirksville, Mo.

American Osteopathic College of Proctology, annual meeting, Hotel Statler, Dallas, Tex., March 27. Secretary, John W. Orman, 1623 E. 15th St., Tulsa 14, Oklahoma.

American Osteopathic College of Radiology, annual meeting, Hotel Statler, Washington, D.C., October 30-November 3. Program Chairman, A. G. Reed, 212 Pythian Bldg., Tulsa 3, Okla. Secretary, F. A. Turfler, Jr., South Bend Osteopathic Hospital, 118 S. William St., South Bend 2, Indiana.

American Osteopathic Hospital Association, annual meeting, Hotel Statler, Washington, D.C., October 31-November 2. Program Chairman, Mrs. Alixe P. Nuzum, Des Moines General Hospital, Des Moines, Iowa. Executive Secretary, Mr. R. P. Chapman, 1013 Kahl Bldg., 326 W. Third St., Davenport, Iowa.

American Osteopathic Society of Proctology, annual meeting, Hotel Statler, Dallas, Texas, March 28-30. Program Chairman, E. R. Horton, Jr., 214 E. DeWald St., Fort Wayne 5, Indiana. Secretary, Carl S. Stillman, Jr., 3523 Fifth Ave., San Diego 3, California.



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British Columbia: See Northwest Osteopathic Convention.

California, annual meeting, Mission Inn, Riverside, May 24-27. Program Chairman, Milton Futterman, 2028 E. First St., Los Angeles 22. Executive Secretary, Mr. Thomas C. Schumacher, 1298 Wilshire Blvd., Los Angeles.

Canadian Osteopathic Association, annual meeting, Mount Royal Hotel, Montreal, October 27-29. Program Chairman, A. E. Wilkinson, 616 Medical Arts Bldg., Montreal 25. Secretary, Miss Joyce C. Currie, 609 Medical Arts Bldg., Montreal 25.

Colorado, Rocky Mountain Conference, Hotel Broadmoor, Colorado Springs, November 11-13. Annual meeting, Denver, May 5. Program Chairman for both meetings, A. E. Nichols, 1332 Del Mar Parkway, Denver 8. Secretary, C. Robert Starks, 1459 Ogden St., Denver 18.

Florida, annual meeting, Fort Harrison Hotel, Clearwater, May 17-19. Program Chairman, Paul E. Wilson, 802 E. Ocklawaha Ave., Ocala. Secretary, Dominic Raffa, 5009 Central Ave., Tampa 3.

Idaho: See Northwest Osteopathic Convention.

Indiana, annual meeting, Notre Dame University and Morris Inn, South Bend, May 14-16. Program Chairman, A. F. Kull, South Bend Osteopathic Hospital, 118 S. William St., South

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Bend 2. Secretary, Arabelle Baker Wolf, 809-13 Odd Fellows Bldg., Indianapolis 4.

Iowa, annual meeting, Hotel Savery, Des Moines, May 20-22. Program Chairman, Clive R. Ayers, Grant. Secretary, Mr. Herman A. Walter, 200 Walnut Bldg., Des Moines 9.

Kentucky, annual meeting, Brown Hotel, Louisville, October 12-13. Program Chairman, Henry P. Lawrence, Hawesville. Secretary, Harold D. Benteen, 2048 Winchester Ave., Ashland.

Maine, midyear meeting, Eastland Hotel, Portland, December 2-3. Secretary, Roswell P. Bates, 72 Main St., Orono.

Massachusetts, annual meeting, Hotel Somerset, Boston, January 21-22. Program Chairman, Everett L. Pierce, Pleasant St., East Dennis. Executive Secretary, Mrs. Gladys M. Stockdale, 524 California St., Newtonville.

Michigan, annual meeting, Civic Auditorium, Grand Rapids, October 3-5. Program Chairman, Harold W. Laidlaw, McCoy-Laidlaw Clinic, Bad Axe. Executive Secretary, Mr. Harve Lamont Smith, 81 Glendale, Highland Park 3.

Minnesota, annual meeting, Minneapolis, May 3-5. Program Chairman, Constance Idtse, 47 S. Ninth St., Minneapolis 2. Secretary, E. R. Komarek, 301 Granite Exchange Bldg., St. Cloud.

Missouri, annual meeting, Continental Hotel and Municipal Auditorium, Kansas City, September 27-29. Program Chairman, J. Edward Sommers, 6504A Delmar Blvd., St. Louis 5. Executive Secretary, Mr. Paul D. Adams, 325 E. McCarty St., Jefferson City.

New Jersey, midyear meeting, Stacy-Trent Hotel, Trenton, September 18. Annual meeting, Essex House, Newark, March 10-11. Program Cochairmen for both meetings, George W. Northup, 104 S. Livingston Ave., Livingston, and Harry A. Sweeney, 25 S. Illinois Ave., Atlantic City. Executive Secretary, Mr. I. J. Tecker, 21 Hancock St., Riverside.

New York, annual meeting, Hotel Statler, New York City, October 14-15. Program Chairman, Walter Streicker, 7 Plaza St., Brooklyn 17. Secretary, Robert E. Cole, 417 S. Main St., Geneva.

North Carolina, annual meeting, Battery Park Hotel, Asheville, October 20-22. Secretary and Program Chairman, S. Dales Foster, 710 Public Service Bldg., Asheville.

Northwest Osteopathic Convention, Chinook Hotel, Yakima, Washington, June 25-27. Program Chairman, Wilbert B. Saunders, 4730 University Way, Seattle 5.

Ohio, refresher course, Commodore Perry Hotel, Toledo, October 26-27. Program Chairman, William B. Carnegie, 813 The Arcade, Cleveland 14. Annual meeting, Neil House, Columbus, May 7-9. Program Chairman, George O. Hoover, 91 S. Main St., Oberlin. Executive Secretary, Mr. William S. Konold, 50 E. Broad St., Columbus 15.

Oklahoma, annual meeting, Hotel Tulsa, Tulsa, November 8-10. Program Chairman, Carl R. Samuels, S. Adair St., Box 44, Pryor. Executive Secretary, Mr. Walter L. Gray, 210-12 Braniff Bldg., Oklahoma City.

Oregon: See Northwest Osteopathic Convention.

Pennsylvania, annual meeting, Bellevue-Stratford Hotel, Philadelphia, September 30-October 2. Program Chairman, George S. Esayian, 113 N. Ambler St., Quakertown. Executive Secretary, Mr. George W. Thomas, 1941 Market St., Harrisburg.

Rhode Island, annual meeting, Warwick Country Club, Warwick, March 7-8. Program Chairman, Richard Dowling, 1039 Broad St., Providence 5. Secretary, J. Weston Abar, 824 Broad St., Providence 7.

Rocky Mountain Conference: See Colorado.

Texas, annual meeting, Baker Hotel, Dallas, April 22-28. Program Chairman, Robert J. Brune, 1819 S. Brownlee, Corpus Christi. Executive Secretary, Phil R. Russell, 512 Bailey St., Fort Worth 7.

Vermont, annual meeting, Long Trail Lodge, Mendon Mt., Rutland, September 24-25. Program Chairman, Hermon K. Sherburne, Jr., Mead Bldg., Rutland. Clerk, Howard I. Slocum, Battell Block, Middlebury.

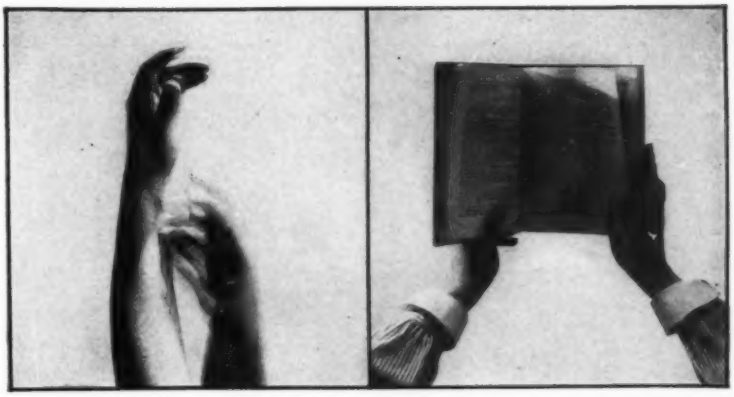
Virginia, midyear meeting, Hotel John Marshall, Richmond, October 8. Annual meeting, The Lodge, Williamsburg, May 25-26. Program Chairman,

Vincent H. Ober, 407-11 Bankers Trust Bldg., Norfolk 10. Secretary, John A. Cifala, 2778 N. Washington Blvd., Arlington.

Washington, midyear meeting, Washington Athletic Club, Seattle, December 3-4. Program Chairman, Erskine H. Burton, 710 Jones Bldg., Tacoma 2. Secretary, Eugene E. LaCroix, 1747 S. Sheridan Ave., Tacoma 5.
See also: Northwest Osteopathic Convention.

Wisconsin, annual meeting, Hotel Wisconsin, Milwaukee, April 23-25. Program Chairman, R. D. Walling, 709 Oak St., Baraboo. Secretary, Edwin J. Elton, 1518 N. 70th St., Wauwatosa 13.

Of two patients with poison ivy...



one aggravates the dermatitis venenata by vicious scratching; the result: excoriation and infectious eczematoid dermatitis.

the other is not disturbed by itching. The dermatitis venenata is permitted to clear rapidly and without annoying complications.

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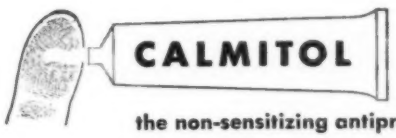

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1. Lubowe, I. I.: New York State J. Med. 50:1743, 1950.

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State and National Boards

ARIZONA

Those interested in professional examinations should contact Russell Peterson, D.O., secretary, Osteopathic Board of Registration and Examination in Medicine and Surgery, 2747 East McDowell Road, Phoenix.

Officers of the Osteopathic Board of Registration and Examination in Medicine and Surgery are: President, Coy L. Purcell, Tucson; vice president, Richard O. McGill, Phoenix; and secretary-treasurer, Russell Peterson, Phoenix.

Basic science examinations September 20 at the University of Arizona, Tucson. Applications must be filed 2 weeks prior to the examination. Address Herbert D. Rhodes, Ph.D., secretary, Basic Science Board, University of Arizona, Tucson.

ARKANSAS

C. W. Dalrymple, Little Rock, has been appointed to the Board of Osteopathic Examiners for a term expiring in 1958.

CALIFORNIA

Vincent P. Carroll, Laguna Beach, and Glen D. Cayler, Los Angeles, have been reappointed to the Board of Osteopathic Examiners for terms expiring December 21, 1957.

COLORADO

Basic science examinations September 7-8 at the Y.M.C.A. Bldg., E. 16th Ave. and Lincoln St., Denver. Applications must be filed on or before August 24. Address Esther B. Starks, D.O., secretary, Basic Science Board, 1459 Ogden St., Denver 18.

CONNECTICUT

Basic science examinations October 8 in Room 23, Lampson Hall, Yale University, New Haven. Applications must be filed 2 weeks prior to examination. Address Miss M. G. Reynolds, executive assistant, Board of Healing Arts, 52 Whitney Ave., New Haven.

DISTRICT OF COLUMBIA

Professional examinations November 14-15 and basic science examinations October 24-25, both at 1740 Massachusetts Ave., N.W., Washington. Applications must be filed by October 1. Address Mr. Paul Foley, Deputy Director, Department of Occupations and Professions, 1740 Massachusetts Ave., N.W., Washington 6.

FLORIDA

Examinations in November. Address R. Philip Coker, D.O., secretary, Board of Osteopathic Medical Examiners, Box 66, Panama City.

IDAHO

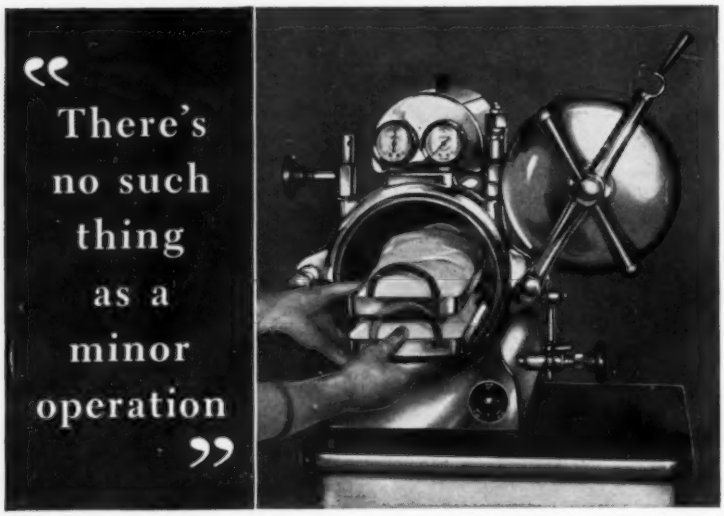
Examinations November 10 at Boise. Address Miss Margaret Gilbert, Director, Occupational License Bureau, Department of Law Enforcement, State House, Boise.

ILLINOIS

Examinations October 11-13 in Chicago. Address Mr. Frederic B. Selcke, Superintendent of Registration, Department of Registration and Education, State House, Springfield.

INDIANA

C. A. Brink, D.O., Princeton, has been elected treasurer of the Board of Medical Registration and Examination.



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IOWA

Basic science examinations October 11 at the Capitol Bldg., Des Moines. Address Ben H. Peterson, Ph.D., secretary, Board of Basic Science Examiners, Coe College, Cedar Rapids.

KANSAS

Officers of the Board of Osteopathic Examination and Registration are: President, Fred Dunlap, Pleasanton; vice president, Courtney Meyers, Madison; and secretary, Forrest H. Kendall, Holton.

C. A. Welker, Concordia, has been reappointed to the Board for a term expiring May 23, 1959.

MAINE

Examinations November 8-9 at Augusta. Address George F. Noel, D.O., secretary, Board of Osteopathic Examination and Registration, Monument Square, Dover-Foxcroft.

MARYLAND

Examinations in October. Address Christopher L. Ginn, D.O., secretary, Board of Osteopathic Examiners, 419 No. Charles St., Baltimore 1.

MICHIGAN

Basic science examinations October 14-15 at Detroit and Ann Arbor. Applications must be filed by October 1. Address Mrs. Anne Baker, secretary, Board of Examiners in the Basic Sciences, 116 Mason Bldg., Lansing.

Emmett Binkert, Carson City, has been appointed a member of the Board of Osteopathic Registration and Examination for a term expiring in 1960.

MINNESOTA

Officers of the Board of Osteopathic Examiners are: President, Robert H. Clark, Northfield; vice president, Edmund C. Goblirsch, Little Falls; and secretary-treasurer, W. F. Kreighbaum, Minneapolis. Phil Morrison, St. Cloud, has been reappointed to the Board for a term expiring in January, 1959.

Basic science examinations October 4-5 at the University of Minnesota, Minneapolis. Applications must be filed by September 10. Address Raymond N. Bieter, M.D., secretary, Board of Examiners in the Basic Sciences, 105 Millard Hall, University of Minnesota, Minneapolis 14.

MISSOURI

E. J. Gahan, Perryville, has been reappointed to the Board of Osteopathic registration and Examination for a term expiring in May, 1960.

MONTANA

Examinations September 6. Address Asa Willard, D.O., secretary, Board of Osteopathic Examiners, Wilma Bldg., Missoula.

NEBRASKA

Basic science examinations October 4-5. Applications must be filed 15 days prior to examination. Address Mr. Husted K. Watson, Director, Bureau of Examining Boards, Department of Health, State Capitol Bldg., Lincoln 9.

NEVADA

Basic science examinations October 4. Address Donald G. Cooney, Ph.D., secretary, Board of Examiners in the Basic Sciences, Box 9005, University Station, Reno.

NEW HAMPSHIRE

Examinations September 14-15 at Concord. Address John S. Wheeler, M.D., secretary, Board of Registration in Medicine, State House, Concord.

NEW JERSEY

Examinations October 18. Address Patrick H. Corrigan, M.D., acting secretary, Board of Medical Examiners, 28 W. State St., Trenton 8.

Edwin T. Ferren, D.O., Camden, has been reappointed to the Board for a term expiring December 18, 1956.

NEW MEXICO

Basic science examinations July 17. Address Mrs. Marguerite Cantrell, secretary, Board of Examiners in the Basic Sciences, P.O. Box 1522, Santa Fe.

NEW YORK

Examinations October 4-7. Address John W. Paige, M.D., chief, Bureau of Professional Examinations and Registrations, 23 S. Pearl St., Albany 7.

OREGON

Basic science examinations September 10. Address Mr. John R. Richards, secretary, State Board of Higher Education, Eugene.

RHODE ISLAND

Professional examinations October 6-

7. Address Mr. Thomas B. Casey, Administrator of Professional Regulation, 366 State Office Bldg., Providence.

Basic science examinations August 31 in Room 366, State Office Bldg., Providence. Applications must be filed approximately 20 days prior to examination. Address Mr. Casey.

TENNESSEE

Examinations in February and July at Nashville. Applications must be filed 15 days prior to examination. Address M. E. Coy, D.O., secretary, Board of Examination and Registration for Osteopathic Physicians, 1226 Highland, Jackson.

Officers and members of the Board of Examination and Registration for Osteopathic Physicians and the expiration of their terms are: President, L. D. Chesemore, July, 1958; secretary, M. E. Coy, July, 1959; Walter Baker, July, 1956; Paul G. Smith, July, 1957; and Harold Roberts, July, 1960. Dr. Roberts is newly appointed, replacing G. A. Bradfute.

TEXAS

Howard R. Coats, D.O., Tyler, has been appointed to the Board of Medical Examiners for a term expiring in 1961.

VERMONT

Officers of the Board of Osteopathic Examination and Registration are: President, H. I. Slocum, Middlebury; secretary, Charles D. Beale, Rutland; and treasurer, J. Harry Spencer, St. Albans. All were re-elected.

Dr. Slocum was reappointed to the Board for a term expiring February 28, 1958.

WEST VIRGINIA

Examinations in October at the Greenbrier Hotel, White Sulphur Springs. Applications must be filed 30 days prior to examination. Address W. S. Irvin, D.O., secretary, Board of Osteopathy, 202 Broadway, Berkeley Springs.

WISCONSIN

Basic science examinations September 23 at the Assembly Chamber State Capitol, Madison. Applications must be filed by September 15. Address Mr. William H. Barber, secretary, Board of Examiners in the Basic Sciences, 621 Ransom St., Ripon.

WYOMING

Examinations October 3 at Cheyenne. Address Franklin D. Yoder, M.D., secretary, Board of Medical Examiners, New State Office Bldg., Cheyenne.

REREGISTRATION OF OSTEOPATHIC LICENSES

Within 60 days after July 1—Indiana, $5.00 for residents, $10.00 for nonresidents. Address Paul R. Tindall, M.D., secretary, Board of Medical Registration and Examination, 538 K. of P. Bldg., Indianapolis 4.

September 1—Ohio, $2.00. Address H. M. Platter, M.D., secretary, State Medical Board, 21 W. Broad St., Columbus 15.

During September—Nebraska, $2.00. Address C. Eugene Brown, D.O., secretary, Osteopathic Board of Examiners, 1702 Second Ave., Nebraska City.

October 31—Pennsylvania, $5.00. Address Mrs. Katherine M. Wollet, acting secretary, Bureau of Professional Licensing, Harrisburg.

On or before November 1—Missouri, $2.00. Address F. C. Hopkins, secretary, Board of Osteopathic Registration and Examination, 205 N. Fourth St., Hannibal.

By November 1—Rhode Island, $1.00. Address Mr. Thomas B. Casey, Administrator of Professional Regulation, 366 State Office Bldg., Providence.

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1. Pomeranze, J. et al. Angiology, June, 1955.
2. Freedman, L. Angiology 6:52, Feb. 1955.
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EXAMINATION BY NATIONAL BOARD

The National Board of Examiners for Osteopathic Physicians and Surgeons conducts Parts I and II of its examination on the first Thursday and Friday of each May and December at the six approved colleges. Application blanks may be obtained from the secretary or the dean of the college, and the completed application blank, together with a passport photograph and check for the parts to be taken, must be in the secretary's office by the November 1 or April 1 preceding the examination.

Examinations in Part I consist of anatomy, including histology and embryology; physiology; physiological chemistry; general pathology; and bacteriology, including parasitology and immunology.

Part II consists of examinations in surgery, including applied anatomy, surgical pathology, and surgical specialties; obstetrics and gynecology; pediatrics; neurology and psychiatry; public health, including hygiene, medical jurisprudence; osteopathic principles, therapeutics, including pharmacology and materia medica.

Part III is an oral and practical examination given under the supervision of a chief examiner who is a member of the Board and by a panel of associate examiners. Subjects covered in Part III are anatomy; physiology; pathology; osteopathic principles; therapeutics, and pharmacology; surgery; ophthalmology and otorhinolaryngology; obstetrics and gynecology; physical and clinical diagnosis; public health and communicable diseases.

These are oral examinations which the candidate may take after having satisfactorily completed the first 6 months of a 1-year internship in a hospital approved by the American Osteopathic Association for intern training. Part III is given annually at the above-named colleges.

Eligibility requirements are as follows: Part I, satisfactory completion of the first 2 years in an approved school of osteopathy; Part II, satisfactory completion of Part I and of the first two quarters or trimesters of the senior year in an approved osteopathic college; Part III, satisfactory completion of Part II and at least 6 months of a 1-year internship approved by the American Osteopathic Association. The internship requirement does not apply to candidates who took Part I prior to July, 1950.

Application must be filed with the secretary of the Board not less than 30 days prior to the examination dates. Address Paul van B. Allen, D.O., secretary, 1512 N. Delaware Street, Indianapolis 2, Indiana.

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PUBLIC CONTROL OF RADIATION EMITTERS*

By Gerald L. Hutton, LL.B.†

Shortly after the discovery of X-ray and the isolation of radium at the turn of the century, radiation was found to be a useful research tool and to offer promises of high utility in the fields of medicine and industry. The potentially harmful properties of X-rays and naturally occurring radioactive materials, such as radium and thorium, were also recognized at an early date. Pierre Curie, H. Becquerel, E. Grubbe, and other scientists reported cases of dermatitis, epilation, and other types of damage from X-rays and injuries traceable to radiation from radium prior to 1900.

Nevertheless, as X-rays were used in World War I for industrial radiographic purposes, only minimal precautions were observed in many instances. The equipment itself did not possess the safety features found on modern X-ray units. During the early 1920's occurred the radium poisoning cases in which a number of girls engaged in painting watch dials apparently ingested pernicious quantities of radium. Reportedly, at least 20 of a total of 800 employees died during subsequent years with symptoms strongly suggestive of radium necrosis or radium-induced tumors. During this same period radium was found to be a useful external and interstitial source of radiation in therapy. Radium was also injected into patients for a variety of ailments ranging from arthritis to gout, and radium water was advertised and sold as a tonic and a rejuvenator.

As early as 1913, the National Bureau of Standards undertook the standardization of radium for medical uses, and in 1929 it sponsored the formation of the Advisory Committee on X-ray and Radium Protection. This committee, the name of which was later changed to the National Committee on Radiation Protection, has prepared a number of handbooks dealing with means of protection against the harmful effects of radiation. They cover such subjects as the safe handling of radioisotopes, control and removal of radioactive contamination in

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laboratories, maximum permissible concentrations in air and water, and disposal of radioactive wastes. The committee is comprised of representatives from such organizations as the Atomic Energy Commission, the Public Health Service, the National Bureau of Standards, the American Medical Association, the Radiological Society of North America, the American Radium Society, and the National Electrical Manufacturers Association. Its recommendations are accepted as reliable and authoritative, and they are the basis for many of the present radiological safety procedures.

With the development of the atomic energy program after 1942, the need for increased attention to the hazards of radiation became apparent. Today, not only substantial amounts of naturally occurring radioisotopes, such as radium and polonium, and approximately 125,000 X-ray units (*1*), but also relatively large quantities of reactor-produced radioisotopes, made available through this program, are in use in medicine, industry, and research. From August 1946, when distribution was begun, through May 1954, the Atomic Energy Commission distributed reactor-produced radioisotopes to over 2,000 medical institutions, physicians, colleges, universities, industrial firms, Federal and State laboratories, foundations, and institutes (*2*). (The actual number of persons currently using radioisotopes is considerably greater than this figure, inasmuch as an institutional program usually includes several persons qualified to handle radioisotopes.) Users of these materials are found in every State, but the greatest number, in order of decreasing representation, are in New York, California, Pennsylvania, Illinois, Ohio, Massachusetts, Texas, New Jersey, Michigan, and Maryland.

The problem of protection against the hazards associated with radioactive materials, both those occurring naturally and those produced artificially, and radiation-generating devices can best be met through the cooperation of the several Federal and State agencies concerned with the protection of the health of the public. The following presentation outlines the functions of each of these agencies in assuring that radiation emitters are handled in a safe manner and indicates how they are interrelated.

AEC'S AUTHORITY AND FUNCTIONS

In the Atomic Energy Act of 1946, the Atomic Energy Commission was authorized to distribute, in accordance with certain general provisions, the byproduct materials (radioisotopes) resulting from operation of nuclear reactors and to establish radiological health safety standards governing the possession and use of these materials. The commission's authority in this regard was expressed with greater precision and clarity in the Atomic Energy Act of 1954. Section 81 of the act provides as follows:

> No person may transfer or receive in interstate commerce, manufacture, produce, transfer, acquire, own, possess, import, or export any byproduct material, except to the extent authorized by this section or by section 82. The Commission is authorized to issue general or specific licenses to applicants seeking to use byproduct material for research or development purposes, for medical therapy, industrial uses, agricultural uses, or such other useful applications as may be developed. The Commission may distribute, sell, loan, or lease such byproduct material as it owns to licensees with or without charge. . . . Licensees of the Commission may distribute byproduct material only to applicants therefor who are licensed by the Commission to receive such byproduct material. The Commission shall not permit the distribution of any byproduct material to any licensee, and shall recall or order the recall of any distributed material from any licensee, who is not equipped to observe or who fails to observe such safety standards to protect health as may be established by the Commission or who uses such material in violation of law or regulation of the Commission or in a manner other than as disclosed in the application therefor or approved by the Commission. The Commission is authorized to establish classes of byproduct material and to exempt certain classes or quantities of material or kinds of uses or users from the requirements for a license set forth in this section when it makes a finding that the exemption of such classes or quantities of such material or such kinds of uses or users will not constitute an unreasonable risk to the common defense and security and to the health and safety of the public.

Section 161 (b) authorizes the commission to:

> Establish by rule, regulation, or order, such standards and instructions to govern the possession and use of special nuclear material, source material, and byproduct material as the Commission may deem necessary or desirable to promote the common defense and security or to protect health or to minimize danger to life or property.

Distribution Regulations

The Federal regulations governing distribution of AEC-controlled radioisotopes are published in the Federal Register and appear also in the Code of

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Federal Regulations, 1953 supplement, title 10, chapter 1, part 30. These regulations set forth the application procedure for obtaining the radioisotopes and limit procurement, possession, and use of the materials, except exempted quantities, to persons holding a valid authorization (license) from the commission.

Exempt from the requirements concerning procurement, possession, and use of radioisotopes are 1 microcurie or less of beta and/or gamma emitters with half lives greater than 30 days and 10 microcuries or less of beta and/or gamma emitters with half lives no greater than 30 days. These quantities, however, may not be combined or otherwise altered or treated so as to increase the rate of radiation exposure above the original radiation intensity, nor may they be used for medical purposes except upon authorization of the commission. In addition, the commission may exempt specific items, upon application for exemption, from any or all of the regulatory requirements.

All persons desiring to procure radioisotopes, other than the exempted quantities, produced in or distributed by or through AEC facilities, produced in any nuclear reactor within the United States, or produced in foreign nuclear reactors for shipment into the United States must file an application for radioisotope procurement, form AEC-313, with the AEC's Isotopes Division, Oak Ridge, Tenn. The technical staff of the Isotopes Division reviews the application to determine if the applicant is equipped to use the requested radioisotope as outlined in the proposed use in a manner that will not endanger health and safety. This review entails an analysis of the applicant's experience and training, facilities, equipment, and radiological safety procedures. Applications involving novel or relatively abstruse medical uses are referred to the AEC's medical advisers for evaluation and recommendation.

If a review of the application indicates that the applicant has the facilities and qualifications necessary to use the requested material in a safe manner, an authorization for radioisotope procurement is issued to the applicant authorizing him to procure, possess, and use the specific type and quantity of radioisotope for which he has applied. If an applicant does not possess sufficient experience in the use of radioactive materials, he is requested to obtain such experience or associate himself with a person possessing the requisite experience and training.. If facilities, equipment, or safety procedures are inadequate, the deficiency must be corrected before an authorization is issued.

Each person possessing or using radioisotopes must keep permanent records showing the receipt, use, storage, delivery, and disposal of radioisotopes, and the safety procedures to protect health. These records must be made available to the commission upon request. Such persons must also permit the commission at all reasonable times to make inspections of facilities where radioisotopes are stored or used.

If the commission at any time finds that a person is not equipped to observe or has failed to observe health safety standards established by the commission, or has used the radioisotopes in a manner other than as stated in his application, or has used the radioisotope in violation of law or any regulation of the commission, the commission may withhold or recall radioisotopes from such person.

Visitation Program

In accordance with the regulation concerning inspections, the Isotopes Division of AEC maintains a staff of technical specialists who visit institutions possessing radioactive materials. They note and evaluate such items as availability of handling equipment; adequacy of laboratory facilities, shielding, ventilation, and storage; radioisotope handling procedures; monitoring instruments and procedures; posting of caution signs; and labeling and waste disposal procedures. They advise on means of improving radiological safety procedures when improvement is indicated.

More than 500 institutions and individual radioisotope users are visited annually by AEC representatives. The visitation program is constantly expanding to meet the problems created by increasing use of radioisotopes. Selection of institutions to be inspected is usually dependent on amounts and types of radioisotopes which the user has received. As a rule, those institutions which possess

large quantities of long-lived radioisotopes, such as strontium-90 and cobalt-60, are visited more frequently than users having only limited amounts of short half-life or low-energy emitters.

These visits to radioisotope users are not only instrumental in improving radiological safety procedures, but they also provide information of value in reviewing future applications from the user. The visits also keep the AEC Isotopes Division informed on specific problems facing radioisotope users in their day-to-day operations.

(To be concluded in the September JOURNAL*)*

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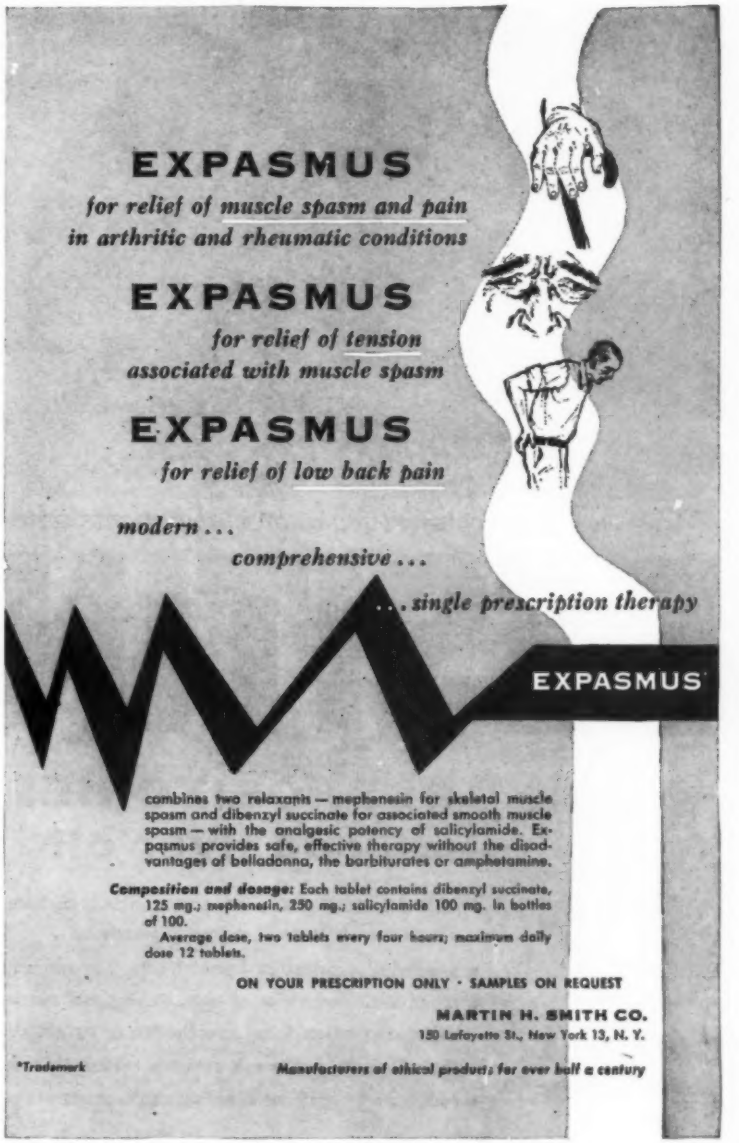
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THE INCREASING CHANCES OF SURVIVAL*

The continuing decline in mortality since the turn of the century has brought better chances of survival to each successive generation of Americans. While the gains have been particularly impressive in early life, substantial progress has also been made at the ages when most men are gainfully employed and when most women bear responsibility for the care of a home and children.

About three fifths of the men who had attained age 20 in 1910 (the people born in 1890) have survived to celebrate their 65th birthday in 1955. The prospect of living through the main working ages of life was not quite as favorable for those born at earlier dates. Thus, of the men born in 1880, 57 percent survived from ages 20 to 65. Judging from available statistics, the corresponding figure was only about one in every two for the men born prior to 1860.

The chances of survival have also improved appreciably at midlife. For example, the proportion surviving from age 40 to 65 increased from 63 percent for the men born in 1860 to 68 percent for those born in 1890. Although the gains were smaller toward the end of productive life, at each attained age the proportion who reached age 65 was somewhat greater for the men born around 1890 than for earlier generations.

A higher proportion of women than men live to the older ages, reflecting the lower mortality normally experienced by women and the fact that they are not generally exposed to the hazards of war. Moreover, their advantage over men with respect to longevity has increased with each new generation. Sixty-eight percent of the women who attained age 40 in 1900 survived to age 65, compared to 63 percent for the corresponding group of men. Of the women reaching age 40 in 1930, 79 percent lived to celebrate their 65th birthday; the comparable figure for males was 68 percent, the record established by women three decades earlier.

The outlook for living beyond age 65 has also improved somewhat since 1900, although the gains for white men have not been continuous. Nevertheless, judging from past experience, most men still have many years of life ahead of them after the usual retirement age. Thus, of the men reaching age 65 in 1935, 79 percent survived for at least 5 years, 57 percent for at least 10 years, and more than 18 percent for at least 20 years. Women have fared even better. In fact, out of the group that attained age 65 two decades ago, five sixths lived to age 70 or beyond and one fourth lived to celebrate their 85th birthday.

Our younger generations will undoubtedly have a more favorable record of survival than persons born before the turn of the century, because death rates have been decreasing since 1900 and are likely to decline still further in the future. However, even if current levels of mortality were to remain unchanged, better than two out of every three men over 20 years of age, and more than four out of every five women at these ages,

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would survive beyond their 65th birthday. How much more favorable the actual record will turn out to be will depend upon many factors, among them further advances in the medical sciences and the rapidity with which they are extended to all segments of the population. Appreciable gains might also occur among men at the older ages, for whom mortality rates have thus far shown relatively little tendency to change.

These trends in longevity have important social and economic implications for our country. In the years ahead, more and more of the population will live to the age of retirement and beyond. At the same time, an increasing proportion of wives will outlive their husbands.

PREMATURITY AND RETROLENTAL FIBROPLASIA*

Herman E. Hilleboe, M.D.
Commissioner, New York State Department of Health

After twelve years of intensive research, it is now clear that premature infants exposed to high concentrations of oxygen are prone to develop blindness. The damaging effects of too much oxygen on the budding retinal vessels of the eye result in a condition known as retrolental fibroplasia (RLF), characterized by scar-tissue behind the lens of the eye. Apparently mature blood vessels are not affected; those in the under-developed retina of the premature infant appear to be sensitive to concentrations of oxygen over 40 per cent. The hazard is greatest when oxygen is given for long periods of time during those critical first few days after birth when the infant is fighting to keep alive.

Regardless of the exact mechanism that produces RLF, restriction of the use of oxygen on prematures markedly reduces or prevents the development of RLF. From a public health viewpoint the widespread application of this knowledge appears to be sufficient to prevent blindness from RLF.

There is no longer any excuse for the routine use of oxygen on every premature infant in the first days of life. The elimination of routine oxygen administration does not decrease the chance of survival of prematures. Controlled studies have shown this to be true. Only if there is definite cyanosis or genuine respiratory dysfunction should oxygen be given. Oxygen should be given under expert medical supervision. Hospital authorities also have a responsibility to assure themselves that no concentrations over 40 per cent are administered, that efficient apparatus is available, and that the nursing staff makes frequent tests with oxygen analyzers to make certain that prematures are receiving correct concentrations. Furthermore, oxygen should be withdrawn from use just as soon as the infant can get along without it.

To be sure, there are many hospitals in New York State that have been following these procedures for some time. We are proud of their leadership and good work in preventing RLF. However, a total of 170 cases of blindness due to RLF has been reported to the Commission for the Blind in New York State. Among hospitals where these cases occurred, the staffs must concentrate on health education and training to prevent blindness from this cause.

It is urgent that we apply on a statewide basis, hospital by hospital, the sight-saving knowledge that proper use of oxygen among prematures can prevent RLF blindness. We will, of course, follow-up all cases of RLF in babies reported to the Commission for the Blind. In this way, we can alert the hospital staffs to the danger of over-exposure of prematures to oxygen, and help them avoid future occurrences. The Commission for the Blind is cooperating wholeheartedly in this follow-up program.

Every case of blindness prevented saves an individual from a lifetime of darkness, not to mention the avoidance of heartache and distress in the parents of a blind child. In addition, such prevention will save the State thousands of dollars for special training and financial aid to the blind.

Once blindness from RLF has occurred, there is nothing that can be done to restore sight. As yet there is no known treatment. Prevention of disease or disability is the shining light of public health; we have before us an unusual

*Editorial reprinted by permission from Health News, New York State Department of Health, May, 1955.

opportunity to avoid the disaster of RLF at its source. Pediatricians, public health physicians and hospital administrators should enlist in the program to eliminate RLF among our premature infants so that these babies may have the chance to go through life with eyes that see.

FUTURE POPULATION TRENDS*

The population of the United States has been increasing steadily and is expected to continue its vigorous growth in the next two decades. Since the last census was taken five years ago, our population, including the Armed Forces overseas, has increased by approximately 13½ million and now totals more than 164½ million. If the birth rate continues at its present high level, the population of our country will pass the 200 million mark before 1970 and will exceed 220 million by 1975. Such increase in the next 20 years would be equal to the gain in the preceding third of a century.

Currently, there are almost 60,000,000 people in our country under age 20 and nearly the same number at ages 20-44; somewhat more than half that number—about 33,000,000—are in the age range 45-64, and not far from 14,000,000 are at ages 65 and over. Each of these groups will increase, with the most rapid growth expected at the younger and the older ages. By 1975, the population at ages 5-19† and at 65 and over will be about 70 percent above that in 1950, according to the Bureau of the Census projections. At ages 45-64 the corresponding increase is expected to be about 40 percent and to exceed 25 percent at ages 20-44.

These varying rates of increase would naturally bring about a change in the age composition of the population. The proportion at ages 65 and over is expected to rise to 9.4 percent of the total population by 1975; the number then would exceed 20½ million, compared with somewhat over 12 million in 1950. It is likewise estimated that the population at the ages under 20 will constitute a somewhat greater part of the total in 1975 than in 1950. In contrast, the proportion at ages 20-44 will decrease, while that at ages 45-64 will remain virtually unchanged.

The sex ratio as well as the age composition of the population is expected to change in the years to come. Females outnumbered males for the first time in the Census of 1950, but by only 1 percent. This margin, it is estimated, will grow to 3 percent by 1975. This trend will reflect the rapid rise in the excess of females over males at the ages 45 and over. At ages 45-64, females will exceed males by more than 10 percent in 1975; at ages 65 and over, the excess will be about 40 percent.

In 1954, our labor force, including the Armed Forces overseas, averaged 68 million. Under conditions of high employment the number may grow to about 72 million by 1960 and to well over 90 million by 1975. While the labor force is growing, its sex and age composition is likely to change somewhat. Women are expected to constitute an increasing part of the labor force. The ratio of females to males may be 43 to 100 in 1960 and 50 to 100 by 1975. Another change in the offing is a moderate increase in the proportion of younger people in the labor force. The proportion at ages 14-24 may show an increase from 19 percent to somewhat over 23 percent of the total between 1960 and 1975.

The West will continue to lead the country in population growth during the next decade, according to projections by the Bureau of the Census. By 1965, California with a population approaching 18 million is expected to have more inhabitants than any other State, outranking New York by a narrow margin. Very largely as a result of this rapid growth in California, the proportion of the country's population in the Pacific States may increase from 10.3 percent at present to 12.1 percent in 1965. Florida, too, is expected to continue adding large numbers to its population, and to reach the 5 million mark in about a decade.

*Reprinted from *Statistical Bulletin*, Metropolitan Life Insurance Company, March, 1955.

†The projections of populations at the younger ages depend upon the future course of the birth rate. The reliability of these projections will be affected by any significant differences between the assumed future birth rates and those actually experienced.

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RECENT TRENDS IN LEUKEMIA*

Only a few decades ago deaths attributed to leukemia, a malignant disease involving the blood and blood-forming organs, were relatively rare, but they have since become an appreciable item in the total mortality from cancer. In 1953, leukemia was responsible for more than 10,000 deaths in the United States; this was about twice the mortality from cancer of the buccal cavity and three times that from skin cancer.

The data based on the experience among persons at ages 1-74 years insured under Industrial policies in the Metropolitan Life Insurance Company show that the death rate rose from 1.8 to 4.7 per 100,000 between 1930 and 1951, but then decreased somewhat. It is difficult to say what the experience of the past few years portends: whether the leukemia mortality has reached the level at which it will become stabilized, or whether the mortality will continue upward after a temporary interruption, as has happened in the past.

Both sexes among these insured experienced a rise in the mortality from leukemia, but the upward trend has been more rapid among males than among females. For white males the age-adjusted death rate more than doubled between 1930-1934 and 1950-1954, while for white females it increased about 80 percent. As a result, the excess mortality among males as compared with that among females increased from nearly 15 percent to more than 35 percent.

Each age group in both sexes recorded an increase in the leukemia death rate between 1930-1934 and 1950-1954. In general, the largest rise occurred among children and among older people—the age groups with the highest mortality. Currently, the death rates are relatively high among preschool children, decrease to a minimum at ages 20-24, and then rise steadily with advance in age. The maximum death rates are recorded at the older ages, but the mortality at ages 1-4 is not exceeded until ages 55-64.

In 1950-1954, leukemia accounted for approximately half of all the deaths from cancer among insured children under 15 years of age. The proportion decreased fairly rapidly with advance in age to one third of the total cancer mortality at ages 15-19, to about one tenth at ages 25-34, and to only 3 percent in the age range 45-74. At every age group, leukemia has been responsible for a larger proportion of all cancer deaths in recent years than 20 years earlier.

The type of leukemia which occurs in childhood is generally different from that at the older ages. In children the disease usually appears in an acute form, and survival from onset is measured in weeks or months. Among older people the chronic form of the disease is the more common, and the duration of life from known onset averages about three years, but may be as much as 10 or more years.

The very rapid fatal course of leukemia among children is pointed up by morbidity data on the disease derived from surveys in the general population of 10 major cities during 1947 and 1948.† At ages under 15 the annual number of deaths from leukemia was two thirds of the total of all known cases at these ages, while at ages 45 and over the proportion was less than one half.

While the statistical record for leukemia makes a somber picture, it is not as dark as it appears at first glance. A large part of the long-term increase in the death rate from the disease undoubtedly reflects merely the more frequent detection of the condition through improved methods of diagnosis. This viewpoint is supported by the fact that the *recorded* death rate from leukemia is highest in those groups of the population which presumably have access to the best diagnostic facilities. Thus, the Northern and the Far Western areas of the United States, where medical facilities are the most developed, generally show the highest leukemia mortality. Similarly, studies of mortality by social class in England and Wales clearly indicate a greater leukemia death rate in the highest socio-economic groups than in the lowest.

Some progress has been made in the treatment of leukemia, particulariy the acute form, through the use of hormones and chemicals. Continued, intensive research on the nature of the disease and the means for its control holds out tangible hopes for the future.

*Reprinted from *Statistical Bulletin*, Metropolitan Life Insurance Company, March, 1955.

†Preliminary summary by American Cancer Society of data from *Cancer Morbidity Series*, 1-10, National Cancer Institute.

Books Received

MEDICAL AND PUBLIC HEALTH LABORATORY METHODS. Successor to Fifth Edition of Laboratory Methods of the United States Army. Edited by James Stevens Simmons, S.B., M.D., Ph.D. Dr. P.H., S.D. (Hon.), Late Brigadier General, U. S. Army (Retired); Dean and Professor of Public Health, Harvard School of Public Health; Former Chief, Preventive Medicine Service, Office of the Surgeon General, U. S. Army, 1940-1946; and Cleon J. Gentzkow, M.D., Ph.D., Colonel, U. S. Army (Retired); Director, Bureau of Laboratories, Department of Health, Commonwealth of Pennsylvania; Former Chief, Division of Chemistry and Physics, Army Medical School, Washington, D. C., 1932-1936 and 1939-1942. Ed. 6. Cloth. Pp. 1191, with illustrations. Price $18.50. Lea & Febiger, Washington Square, Philadelphia 6, 1955.

TEACHING PHYSIOLOGY AND ANATOMY IN NURSING. Signposts for Science Teachers. By Hessel H. Flitter, R.N., B.S., M.A., Assistant Professor and Consultant on Curriculum and Evaluation, School of Nursing, University of Pennsylvania; and Harold R. Rowe, R.N., B.S., M.S., Assistant Professor and Advisor to the Hospital Contract Program (Central Science Teaching Service), School of Nursing, University of Pennsylvania. Cloth. Pp. 56. Price $2.00. J. B. Lippincott Company, East Washington Square, Philadelphia 5, 1955.

MIGRAINE AND PERIODIC HEADACHE. A Modern Approach to Successful Treatment. By Nevil Leyton, M.A., (Cantab.), M.R.C.S., L.R.C.P., Honorary Physician, Migraine Clinic, Putney Health Centre; Clinical Assistant, King Edward VII Hospital, Windsor. Ed. 2. Cloth. Pp. 128. Price $2.50. William Heinemann Medical Books, London. Charles C Thomas, 301-327 E. Lawrence Ave., Springfield, Ill., 1954.

PRACTITIONERS' CONFERENCES Held at The New York Hospital-Cornell Medical Center. Vol. 1. Edited by Claude E. Forkner, M.D., F.A.C.P., Professor of Clinical Medicine, Cornell University Medical College; Attending Physician, The New York Hospital. Cloth. Pp. 411. Price $6.75. Appleton-Century-Crofts, 35 West 32nd St., New York 1, 1955.

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Cutler, Hilton L., (Renewal) 1233 N. La Brea Ave., Los Angeles 38
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1. Cass, L. J. and Frederik, W. S.: Malt Soup Extract as a Bowel Content Modifier in Geriatric Constipation. Journal-Lancet, 73:414 (Oct.) 1953.

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Abbott, Edward T., from 1385 E. Colorado Blvd., to 1601 Capistrano Ave., Glendale 8, Calif.

Adland, Leonard, from 4133½ E. Gage Ave., to 4135 E. Gage Ave., Bell, Calif.

Adler, Keneth, from 2646 Jackson Ave., to Blue Valley Clinic, 5811 Truman Road, Kansas City, 26, Mo.

Alloy, Paul C., from 3008 W. Eighth St., to 3914 Wilshire Blvd., Los Angeles 5, Calif.

Anderson, Edward G., from Box 71, to Box 578, Nucla, Colo.

Angell, E. O., from Moberly, Mo., to 10518 St. Clair Ave., Cleveland 8, Ohio.

Aquila, Michael R., from 1130 Pierce St., to 1441 Ritner St., Philadelphia 45, Pa.

Attarian, Archie B., from Detroit, Mich., to 113 N. Ambler St., Quakertown, Pa.

Baker, Herman A., Jr., from Warren, Ohio, to 16½ W. Broad St., Newton Falls, Ohio

Barnett, Jack E., from Box 431, to Box 221, Bridge City, Texas.

Bastedo, G. C., from 13535 Woodward Ave., to 16 E. Grand Ave., Highland Park 3, Mich.

Bennett, Gerald D., from Boston, Mass., to 214 Lancaster St., Albany 6, N. Y.

Berrey, Ivan Willard, from Oklahoma City, Okla., to 618 E. Hurd St., Edmond, Okla.

Biddison, Eddie I., from Columbus, Ohio, to 510 E. Church Ave., Dade City, Fla.

Black, Arthur R., from Trinidad, Colo., to 4411 Montrose Blvd., Houston 6, Texas

Bland, Coburn C., from Lansing, Mich., to 1298 Haslett Road, Haslett, Mich.

Boehm, Gerhard W., from Los Angeles, Calif., to 2114 Redondo Beach Blvd., Redondo Beach, Calif.

Borzilleri, Frank J., from Harrisburg, Pa., to 2401 Warwick Road, Alhambra, Calif.

Bower, Jean Stommel, from 164 N. Oak Knoll Ave., to Bower-Suffern Medical Group Bldg., 126 N. Oak Knoll Ave., Pasadena, Calif.

Buck, George E., Jr., from Grove City, Pa., to 110 King St., Johnstown, Pa.

Buller, William E., from 1103 Walnut St., to 2625 Liberty St., Allentown, Pa.

Bullis, Harvey R., from 408 First Wisconsin Natl. Bank Bldg., to 735 W. Capitol Drive, Milwaukee 6, Wis.

Caldwell, Neil R., from Dayton, Ohio, to Ivins-Jameson Bldg., Lebanon, Ohio.

Cantor, Harvey, from Los Angeles, Calif., to 8853 Santa Monica Blvd., West Hollywood 46, Calif.

Cavendish, Carl L., from Cuyahoga Falls, Ohio, to 613 Fifth Ave., Montgomery, W. Va.

Chambers, Richard K., Jr., from Lancaster, Pa., to 401 S. Fourth St., Denver, Pa.

Christensen, Norma J., from 3422 N. 22nd St., to 7956 Verree Road, Philadelphia 11, Pa.

Coffey, Lucille M., from 2639 Ninth St., N., to 1401 16th St., N., St. Petersburg 2, Fla.

Colfer, William V., from Norwalk, Conn., to 9 Pine Hill Road, East Norwalk, Conn.

Cummings, Dwain W., from DesMoines, Iowa, to North Ave., Professional Bldg., 170 North Ave., Battle Creek, Mich.

Datzker, Edwin A., COPS '54; 5315 Via San de Larro, Los Angeles 22, Calif.

Doll, Oliver B., from Melcher, Iowa, to 1212 Robinson St., Knoxville, Iowa

Drapkin, Harold, From Los Angeles, Calif., to 17223 Grenshaw Blvd., Torrance, Calif.

Falcone, Robert E., from Ravenna, Ohio, to 35 South Ave., Tallmadge, Ohio

Farquharson, June L., from Cuyahoga Falls, Ohio, to 101 Left Road, Spring Arbor, Mich.

Fischer, John T., Jr., from South Bend, Ind., to Detroit Osteopathic Hospital, 12523 Third Ave., Detroit 3, Mich.

Fiyalko, Boris, from Cleveland, Ohio, to 27521 Lorain Road, North Olmsted, Ohio

Gateman, David, from Miami, Fla., to 2516 Ponce de Leon Blvd., Coral Gables 34, Fla.

Gnau, Charles U., from Fort Myers, Fla., to 134 E. Pasadena Ave., Phoenix 43, Ariz.

Gordon, Irving M., from Detroit, Mich., to 2310 University Ave., N. W., Canton 9, Ohio

Gustafson, Robert W., DMS '55; 337 E. Coast St., Des Moines 13, Iowa

Hall, John D., from 109 E. Williams St., to 125 E. William St., Kendallville, Ind.

Harbaugh, Edward D., from 744 W. Ninth St., to 617 Wright Bldg., Tulsa 3, Okla.

Harker, Bernice, from Los Angeles, Calif., to 6355 Selma Ave., Hollywood 28, Calif.

Harrison, Jim T., from Thomasville, Ga., to Wight Bldg., Cairo, Ga.

Hartwell, Elbert E., from Salt Lake City, Utah, to Route M. R., Emmett, Idaho

Hatch, Loren L., from Jesup, Ga., to Hinesville, Ga.

Heisler, John L., from Kansas City, Mo., to Buckner, Mo.
Heller, Harry A., from 4244 Livernois Ave., to 23738 Joy Road, Detroit 39, Mich.
Hill, James R., from York, Pa., to Broadway, Stewartstown, Pa.
Hindman, Robert J., from Trenton, Mich., to 964 Harding, Plymouth, Mich.
Hirschberg, Herman G., from 2501 Nostrand Ave., to 2400 Nostrand Ave., Brooklyn 10, N. Y.
Hoversten, Lester, from Regensburger St., 15, to c/o Dr. W. Axhausen, Helmstedter St. 8, West Berlin, Wilmersdorf, Germany
Imhoff, Clyde L., Jr., from Toledo, Ohio, to Williston, Ohio.
Irwin, Lowell E., from Alhambra, Calif., to 3914 Wilshire Blvd., Los Angeles 5, Calif.
Jaggers, Donald B., from Kirksville, Mo., to 916 Ocean Ave. Ocean City, N. J.
James, Lester H., from Sylvan Grove, Kans., to 4345 S. Broadway, Wichita 15, Kans.
Jones, J. Bartley, from Friendsville, Tenn., to 6425 Richmond Road, Houston 19, Texas
Junkermann, Richard U., from Columbus, Ohio, to 117 N. Main St., London, Ohio
Kaye, John E., from Comanche, Texas, to Du Bose Bldg., Box 252, Aransas Pass, Texas
Kehoe, Edward P., from 2410 S. MacGregor Way, to 7750 Ley Road, Houston 16, Texas
Kerwood, Robert I., from Fort Worth, Texas, to 606 N. Gray St., Killeen, Texas.
Kirlin, Edmund J., from 549 W. Washington Ave., to Box 675, Council Bluffs, Iowa
Knollhoff, Edwin B., from 3007 N. E. Eighth Ave., to 809 N. Taylor, Amarillo, Texas
Laderman, David R., from 2367 Rustic Road, to 416 W. Norman Ave., Dayton 6, Ohio
Lavell, Louis W., from Kenmore, Wash., to 22327 Marine View Drive, Des Moines, Wash.
Leysack, Alex Eli, from Hermosa Beach, Calif, to 215 Park Ave., Dunsmuir, Calif.
Lloyd, William J., from Allentown, Pa., to 1343 Catasauqua Road, Bethlehem, Pa.
Loest, Edward H., from 164 N. E. 79th St., to 498 E. 78th St., Miami 38, Fla.
Lopat, Stanley P., from Ontario, Calif., to Euclid Ave., at Seventh St., Upland, Calif.
Madziar, Roman J., from Dallas, Texas, to 909 Fitzhugh, Bay City, Mich.
Mahler, Lawrence P., from 406 Ouray Ave., to 1640 U. S. Highway 50, Grand Junction, Colo.
Mahoney, Kenneth J., from Limington, Maine, to 270 Turner St., Auburn, Maine
Maldonado, Robert A., from Azusa, Calif., to 341 S. Heathdale, Covina, Calif.
Marquardt, Roger R., from 765 E. Hamilton Ave., to 1202 E. Atherton Road, Flint 7, Mich.
Marshall, Thomas E., from 31 E. McDowell Road, to 1534 W. Windsor, Phoenix 32, Ariz.
Marx, Bertram H., from Harrisburg, Pa., to G4504 Lapeer Road, Flint 7, Mich.
McDaniel, T. C., from Dublin, Ohio, to 263 Maple Heights, New Lexington, Ohio.
McDowell, William G., from Grove City, Pa., to 412 Dollar Title Bldg., Sharon, Pa.
Meck, Mervin E., from Oak Hill, W. Va., to 210 Canal St., New Smyrna Beach, Fla.
Middleton, Robert L., from 725 Sixth Ave., to 2213 Cottage Grove Ave., Des Moines 11, Iowa
Millay, E. O., from Route 2, to Route 1, Box 78, Noel, Mo.
Miner, William W., from Rutherford, N. J., to East Shore Road, Jamestown, R. I.
Molisky, Albert, from 136 Harmon Ave., to 1412 Pennsylvania Ave., Weirton Heights, Weirton, W. Va.
Mrstik, Lloyd L., from 4834 Yorkshire, to Detroit Osteopathic Hospital, 12523 Third Ave., Detroit 3, Mich.
Mulkin, Marion A., from Kansas City, Mo., to 303 S. Franklin St., Kirksville, Mo.
Murray, Edward C., from Lakewood, Ohio, to Osteopathic Hospital of Kansas City, 926 E. 11th St., Kansas City 6, Mo.
Musial, Matthew A., from Traverse City, Mich., to Burton Heights Osteopathic Hospital, 1922 Division Ave., S., Grand Rapids 7, Mich.
Myers, Harmon L., from Kirksville, Mo., to El Rancho Village Shopping Center, San Jose Estates, Bisbee, Ariz.
November, H. John, from Tujunga, Calif., to 8940 Woodman, Pacoima, Calif.
O'Dell, Vernon F., Jr., from Kansas City, Mo., to 1835 Grand Ave., Grand Junction, Colo.
Olson, Dean R., from Carson City Hospital, to 321 E. Maple, Carson City, Mich.
Orth, H. C., Jr., from 2521 W. Jefferson Ave., to 3340 Edsel Drive, Trenton, Mich.
Owens, Chester R., from Molton, Iowa, to 7609 Grandview, Arvada, Colo.
Parsons, Gordon S., from Westbrook, Maine, to 43 Potter St., Hyannis, Mass.
Payne, David H., from Los Angeles, Calif., to 8345 Talbert Ave., Huntington Beach, Calif.
Payne, William H., from Kirksville, Mo., to Whitaker Osteopathic Hospital, 205 S. Fifth St., Moberly, Mo.
Poppe, Herman E., from 346 Clearbrook Ave., to 147 W. Pumstead Ave., Lansdowne, Pa.
Price, Abraham Leonard, from 169 W. Albanus St., to 2550 E. Allegheny Ave., Philadelphia 34, Pa.
Raffa, Anthony, Jr., from Liberty, N. Y., to 5009 Central Ave., Tampa 3, Fla.
Ratner, Lawrence B., from 1820 Avenue N., to 571 Lefferts Ave., Brooklyn 3, N. Y.
Reed, F. L., from 124 E. Pine, to 807 S. Denver, Tulsa 5, Okla.
Renn, Richard H., from 165 George St., to 2105 West Road, Trenton, Mich.
Rerucha, V. J., from Columbus, Ohio, to 912 Mercer Park Road, Omaha 3, Nebr.
Robinson, William R., from Mount Clemens General Hospital, to 1268 S. Gratiot Ave., Mount Clemens, Mich.
Rogers, H. M., from Columbus, Ohio, to 2470 Riverside, Trenton, Mich.
Roitz, Antone John, from Tulsa, Okla., to Mound City, Kans.
Rosen, Herbert, from Des Moines, Iowa, to Runnells, Iowa
Rossi, Amerigo A., from Whittier, Calif., to 12062 E. Locust St. Norwalk, Calif.
Rothman, David, from Philadelphia, Pa., to Still Osteopathic Hospital, 725 Sixth Ave., Des Moines 9, Iowa
Roulston, George S., from Lansing, Mich., to New Valley Osteopathic Hospital, 3003 Tieton Drive, Yakima, Wash.
Rowlett, Thomas M., Jr., from Tulsa, Okla., to Box 156, Mount Vernon, Texas
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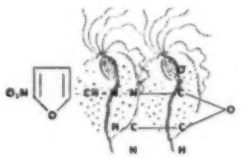

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Smith, Leonard, from Kansas City, Mo., to 78-31 265th St., Floral Park, N. Y.
Smith, Lloyd George, from 1060 N. W. 79th St., to 9946 N. W. 24th Ave., Miami 1, Fla.
Snow, Milton R., from Des Moines, Iowa, to 209 Medical Center Bldg., Medford, Ore.
Stark, Richard J., from Marietta, Pa., to 236 S. Broad St., Lititz, Pa.
Steelman, Joseph E., from York, Pa., to 389 E. Market St., Hellam, Pa.
Steinecker, Henry W., from 211 Cherry Drive, to 173 Cherry Drive, Centerville, Ohio
Steinsnyder, Wynne Alan, from 4814 Larchwood Ave., to 439 S. 46th St., Philadelphia 43, Pa.
Stettner, Max M., DMS '55; Dallas Osteopathic Hospital, 5003 Ross Ave., Dallas 6, Texas
Stiles, Leslie E., from Asbury, Mo., to 110 E. Pennell, Carl Junction, Mo.
Stoll, Selma, from Los Angeles, Calif., to 1192 Ocean Ave., Brooklyn 30, N. Y.
Stover, James D., from 615 David Stott Bldg., to 8646 Puritan Ave., Detroit 38, Mich.
Strickler, Jack Murphy, from 405 Grand Ave., to 5812 N. Main St., Dayton 5, Ohio
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Sunderwirth, C. H., from 100½ N. Main St., to 127 W. Spring St., El Dorado, Springs, Mo.
Surridge, Edgar Donald, Jr., from Mount Clemens, Mich., to Salem-Warren Road, Ohio Route 45, North Jackson, Ohio
Tamez, Richard J., from Columbus, Ohio, to 622 S. Josephine Tobin Drive, San Antonio 1, Texas
Tedrick, C. A., from Jefferson City, Mo., to Phoenix Osteopathic Hospital, 4004 N. Seventh St., Phoenix 43, Ariz.
Thompson, Homer M., from Borger, Texas, to 100 Thomas, Plainview, Texas
Thomsen, Frederick H., from 8635 Artesia Ave., to 9542 E. Artesia Ave., Bellflower, Calif.
Todaro, Emil L., from 9538 N. W. Seventh Ave., to 164 N. E. 79th St., Miami 38, Fla.
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Van De Linder, O. R., from Sallisaw, Okla., to 424 W. Broadway, Enid, Okla.
Varner, Edythe Gates, from Los Angeles, Calif., to 2112 Pine St., Philadelphia 3, Pa.
Vinson, Russell Lee, from Fort Worth, Texas, to 606 N. Gray St., Killeen, Texas
Wagner, Walter P., from Cleveland, Ohio, to Middlefield, Ohio
Walters, Herbert A., from 1005 Jefferson Ave., to 1213 Delaware Ave., Wilmington 6, Del.
Watkins, Dorothy M., from Kansas City, Mo., to 809 W. Lexington St. Independence, Mo.
Weaver, Richman G., from Elkins Park, Pa., to Route 1, Glen Rock, Pa.
White, J. G., from St. Louis, Mo., to Hawk Point, Mo.
Whyte, John A., from Philadelphia, Pa., to 131 Forest St., Bristol, Pa.
Williams, Floyd S., from Denver, Colo, to Scotland, S. Dak.
Williams, W. Ben, from Atlanta, Ga., to Box 546, Tucker, Ga.
Willoughby, Thomas Marsh, from 1816 West St., to 1923 Geneva St., Sioux City 17, Iowa
Witty, Drake R. A., CCO '54; 11124 S. Artesian Ave., Chicago 43, Ill.
Wohlstadter, Jack S., from Los Angeles, Calif., to 8853 Santa Monica Blvd., West Hollywood 46, Calif.
Wong, Ronald Anton, from Seattle, Wash., to 152 W. 64th St., Los Angeles 3, Calif.
Woods, John M., Jr., from 551 Portage Lakes Drive, to 1250 Kenmore Blvd., Akron 14, Ohio
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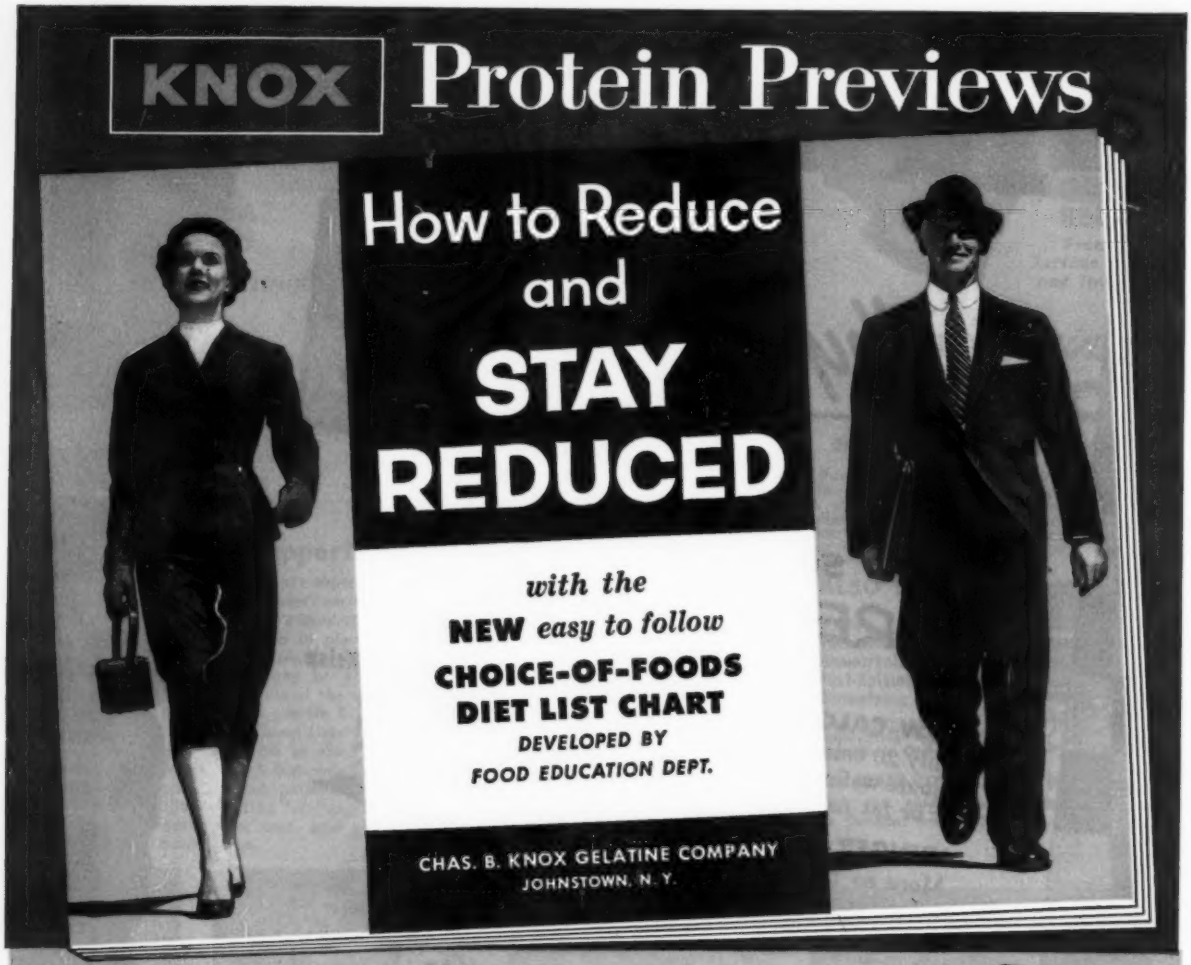
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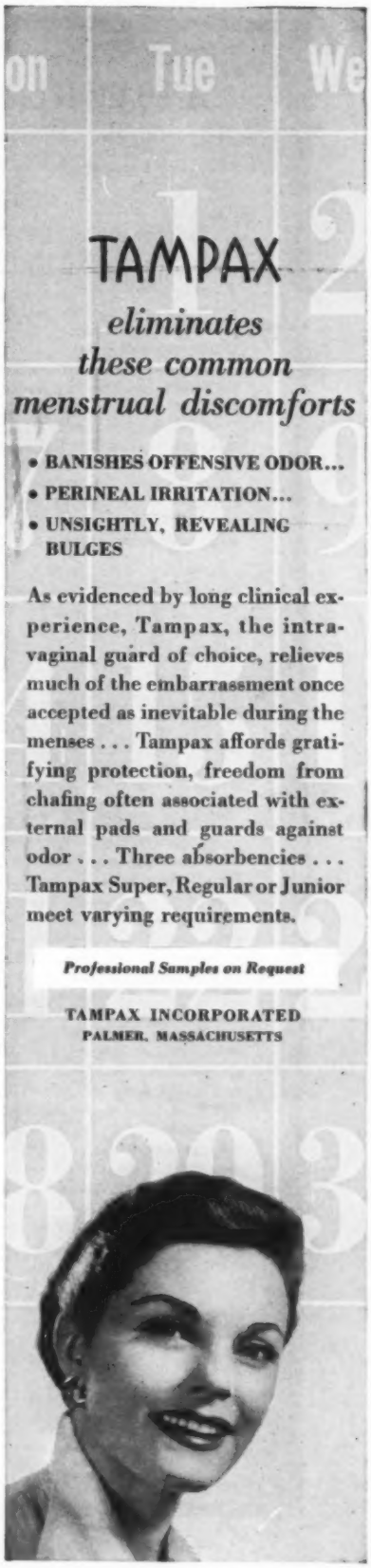
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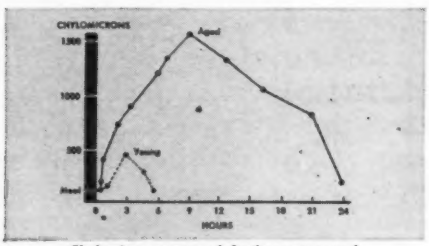
ATHEROSCLEROSIS

Revised concepts of etiology predicate new therapeutic approach

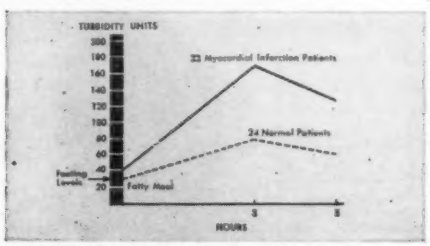
Recent studies attach increasing importance to the particle size and physical characteristics of certain blood lipids, rather than total serum cholesterol as such, in the genesis of atherosclerosis. Assays of neutral fat particles in the blood (chylomicra) following fat ingestion, and the closely related concentration of low-density "giant" lipoprotein molecules, show much greater correlation with clinical atherosclerosis than either the serum cholesterol level *per se* or the cholesterol-phospholipid ratio.

It has also been shown that (1) a high incidence of hypercoagulability and low blood heparin levels exist in patients with cardiovascular disease and atherosclerosis; (2) circulating heparin tends to decrease with age; and (3) an inverse ratio exists between the concentration of giant lipoprotein molecules and serum heparin levels.



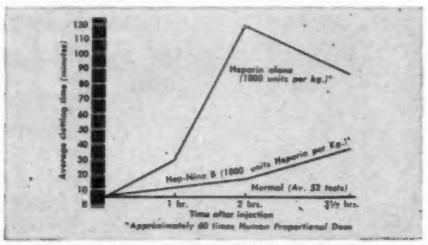
Chylomicron curves of fasting young and old subjects after a Standard fat meal. After Becker et al: Science 110:529, 1949.

Parenterally administered, heparin exerts a profound "clearing" action on chylomicra and the giant molecules. This action is independent of heparin's anticoagulant effect. In the treatment of atherosclerosis, the addition of choline and specific B vitamins appears to enhance heparin's efficacy. Vitamin B_{12} and folic acid aid in the synthesis of labile methyl groups and the transmethylation process. Choline specifically increases the phospholipid turnover, and parenterally administered, it has been shown to have a distinct vasodilating effect. *Most significantly, however, choline decreases the anticoagulant action of heparin, when both drugs are administered simultaneously at the same site, without impairing the clearing effect of heparin.* Thus the use of heparin for atherosclerotic diseases is rendered safe as a routine office procedure, without necessity for periodic clotting time determinations.



Fat Tolerance in Myocardial Infarction and Control Patients. From data of Schwartz et al: JAMA 149:364, 1952.

A preliminary clinical report* on HEP-NINE B —which combines heparin, choline, vitamin B_{12}, folic acid and niacinamide for intramuscular injection—indicates that the combination offers considerable promise in a variety of conditions in which atherosclerosis plays a part, such as angina pectoris, myocardial infarction, coronary disease, related kidney and liver diseases, diabetes mellitus, and certain



Comparison of effects of Hep-Nine B and Heparin alone on clotting time

cases of obesity. Pharmacologic studies showed no significant effect on coagulation time, even in dosage far exceeding that recommended. Chylomicron concentration was reduced promptly in all cases following a single injection, ranging from a minimum 29% reduction (diagnosis: anterior myocardial infarction) to a maximum of 100% (diagnosis: multiple cerebral thrombosis). In patients selected for a history of myocardial infarction or diabetes, the atherogenic index as determined by the Gofman Serum Lipoprotein Test was materially reduced in all cases without benefit of diet restriction. Of 30 patients with recurrent angina pectoris, 23 experienced marked reduction in frequency and severity of episodes. Nitroglycerine requirements were reduced and exercise tolerance was increased in all cases. No patient suffered coronary occlusion or myocardial infarction during the period of study.

Hep-Nine B

Represents a safe office procedure for the treatment of atherosclerosis. Hospitalization and periodic clotting-time determinations are not required. Each cc. contains:

Heparin Sodium (2500 units)	25 mg.
Choline Chloride	100 mg.
Vitamin B_{12}	15 mcg.
Folic Acid	2 mg.
Niacinamide	50 mg.

Recommended dosage is 1 or 2 cc. intramuscularly, once or twice weekly. Supplied in 10 cc. multiple dose vials.

The Columbus Pharmacal Company
Columbus 15, Ohio

Send for complete information and references.

Read, J. T., and Obetz, R. C.: Clinical Experience with Parenteral Heparin-Lipotropic Therapy in Cardiovascular Diseases. Ohio State M. J. (In press).

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Cholecystogram before NEO-CHOLEX administration

Cholecystogram 30 minutes after NEO-CHOLEX administration

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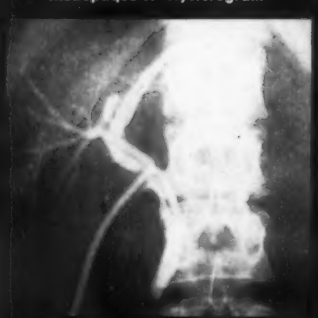

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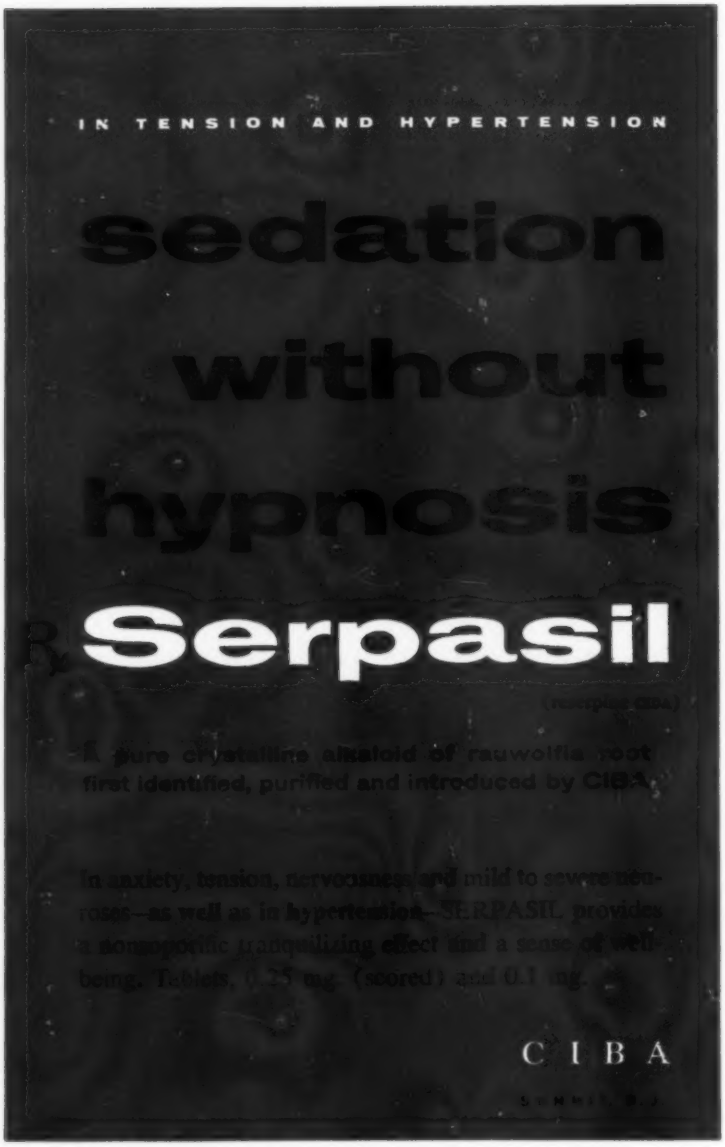

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